In the United States Court of Federal Claims

OFFICE OF SPECIAL MASTERS

No. 14-978V

Filed: April 27, 2022

HARVARD DAVIS,	*	PUBLISHED
Petitioner,	*	
v.	*	Influenza ("Flu") Vaccine; Guillain-Barré Syndrome ("GBS"); Chronic Inflammatory Demyelinating Polyneuropathy ("CIDP").
SECRETARY OF HEALTH AND HUMAN SERVICES,	*	
Respondent.	*	

Lisa Roquemore, Esq., Law Office of Lisa A. Roquemore, Rancho Santa Margarita, CA, for petitioner.
Adriana Teitel, Esq., U. S. Department of Justice, Washington, D.C., for respondent.

RULING ON ENTITLEMENT[1]

Roth, Special Master:

On October 14, 2014, Harvard Davis ("Mr. Davis" or "petitioner") timely filed a petition for compensation under the National Vaccine Injury Compensation Program, 42 U.S.C. § 300aa-10, et seq.[2] (the "Vaccine Act" or "Program"), alleging that the influenza ("flu") vaccination that he received on August 19, 2013 caused him to develop Guillain-Barre Syndrome ("GBS") and chronic inflammatory demyelinating polyneuropathy ("CIDP"). Petition at 2, 7.

For the reasons stated herein, I find that petitioner's evidence is sufficient to demonstrate that the flu vaccine he received on August 19, 2013 was a substantial triggering factor for

[1] This Ruling has been designated "to be published," which means I am directing it to be posted on the Court of Federal Claims's website, in accordance with the E-Government Act of 2002, Pub. L. No. 107-347, 116 Stat. 2899, 2913 (codified as amended at 44 U.S.C. § 3501 note (2006)). **This means the Ruling will be available to anyone with access to the internet.** However, the parties may object to the Ruling's inclusion of certain kinds of confidential information. Specifically, under Vaccine Rule 18(b), each party has fourteen days within which to request redaction "of any information furnished by that party: (1) that is a trade secret or commercial or financial in substance and is privileged or confidential; or (2) that includes medical files or similar files, the disclosure of which would constitute a clearly unwarranted invasion of privacy." Vaccine Rule 18(b). Otherwise, the whole Ruling will be available to the public. *Id.*

[2] National Childhood Vaccine Injury Act of 1986, Pub. L. No. 99-660, 100 Stat. 3755. Hereinafter, for ease of citation, all "§" references to the Vaccine Act will be to the pertinent subparagraph of 42 U.S.C. § 300aa (2012).

"probable CIDP." Due to petitioner's coexisting health issues, this decision also addresses petitioner's comorbidities and how they contribute[d] to his ongoing debility. Accordingly, I find that petitioner is entitled to compensation for those injuries associated with his "probable CIDP".

I. Issues to be Determined

The parties dispute the onset of petitioner's injury following his August 19, 2013 influenza vaccine, whether that injury is CIDP, the contribution of his comorbidities to his alleged CIDP, and all three prongs of *Althen*. Pet. Prehearing Submission, ECF No. 124.

II. Factual Background

A. Procedural History

Mr. Davis filed his petition on October 14, 2014. ECF No. 1. Petitioner filed medical records on numerous occasions: October 15, 2014, ECF Nos. 5-7; November 26, 2014, ECF No. 11; March 16, 2015, ECF No. 17-19; May 20, 2015, ECF No. 25; July 7, 2015, ECF No. 29; August 21, 2015, ECF No. 33; November 9, 2015, ECF No. 39; September 27, 2016, ECF No. 47; January 3, 2017, ECF No. 57; May 24, 2017, ECF No. 70; August 9, 2018, ECF No. 84; August 16, 2018, ECF No. 85; September 27, 2018, ECF No. 87; October 16, 2018, ECF No. 88; November 23, 2018, ECF No. 89; December 4, 2018, ECF No. 91; December 20, 2018, ECF No. 92; January 24, 2019, ECF No. 93; February 19, 2019, ECF No. 98; March 12, 2019, ECF No. 111; March 19, 2019, ECF No. 117; March 21, 2019, ECF No. 120; May 9, 2019, ECF No. 132; May 30, 2019, ECF No. 134; June 24, 2019, ECF No. 135; January 9, 2020, ECF Nos. 147-150; and November 17, 2021, ECF No. 168.

Petitioner filed expert reports from Dr. Steinman with literature on March 30, 2017, ECF No. 64; December 28, 2017, ECF No. 76; August 16, 2018, ECF No. 86; and September 5, 2019, ECF No. 95. Petitioner filed medical literature on March 30, 2017, ECF No. 64; December 28, 2017, ECF No. 76; December 3, 2018, ECF No. 90; February 25, 2019, ECF No. 95; February 27, 2019, ECF No. 100; March 12, 2019, ECF Nos. 103-106; March 14, 2019, ECF No. 112; September 5, 2019, ECF No. 115; September 11, 2019, ECF No.138; and December 16, 2019, ECF No. 140.

Respondent filed expert reports from Dr. Chaudhry with medical literature on September 19, 2017, ECF No. 73; May 14, 2018, ECF No. 80; and February 22, 2019, ECF No. 99. Additional medical literature was filed on March 7, 2019. ECF No. 112.

On April 15, 2015, Respondent filed a status report indicating a willingness to engage in settlement negotiations. The matter proceeded on a settlement track for over 14 months until June 29, 2016, when petitioner filed a status report stating that the parties had reached an impasse in negotiations. ECF Nos. 21, 44. Additional medical records and supplemental expert reports were filed, and the matter then proceeded on a dual track of renewed settlement negotiations and scheduling and preparing for hearing. After attempts at resolution again failed, an entitlement hearing was set for and held on April 4 and 5, 2019 in Sacramento, CA. Despite two full days of hearing, the matter could not be completed and resumed on October 30, 2019 in Washington, DC.

Additional evidence was filed after the hearing was completed in full. Post-hearing briefs were filed by both parties on August 31, 2020, and petitioner filed a reply brief on October 15, 2020. ECF Nos. 165-167. Petitioner filed updated medical records on November 17, 2021. ECF No. 168.

This matter is now ripe for decision.

B. Medical History

1. Petitioner's Health Before Receiving the Influenza Vaccine

Petitioner was born on June 12, 1957. He has a complicated medical history, which includes but is not limited to hypertension, transient ischemic stroke, hypercholesteremia,[3] uncontrolled Type 2 diabetes mellitus ("DM") with peripheral vascular complications and diabetic neuropathy,[4] dysphagia,[5] benign prostatic hyperplasia,[6] stress urinary incontinence, chronic neck, back and hip pain, depression, gastroesophageal reflux disease ("GERD"), anemia, and vitamin D deficiency. Pet. Ex. 16 at 10; Pet. Ex. 89 at 4. Petitioner underwent five spinal surgeries in 1995, 1999, 2000, 2001, and 2002 and the implantation of a spinal cord stimulator, which is reportedly no longer functional but remains in place embedded in tissue. Surgery for its removal was recommended so MRIs of the lumbar spine could be performed due to suspected cord compression, but petitioner opted not to undergo the surgery. Pet. Ex. 10; Pet. Ex. 58 at 2; Pet. Ex. 60 at 3; Pet. Ex. 63 at 2-3.

More specifically, in 2009, petitioner was treated for a host of health issues, including but not limited to chronic lower back pain;[7] intramuscular lipoma[8] with surgical removal;[9] sudden weakness, slurred speech, and stroke;[10] numbness of the face and left leg, memory issues, disorientation, and neurological deficits, though his EKG and head CT were negative;[11] syncope

[3] Hypercholesteremia is another name for hypercholesterolemia, which is defined as "excessive cholesterol in the blood." *Dorland's Illustrated Medical Dictionary* 876. (33rd ed. 2019) [hereinafter "*Dorland's*"].
[4] Neuropathy is "a functional disturbance or pathologic change in the peripheral nervous system, sometimes limited to noninflammatory lesions as opposed to those of neuritis; the etiology may be known or unknown. Known etiologies include complications of other diseases (such as diabetes or porphyria), or of toxicity states [ ]." *Dorland's* 1250. The peripheral nervous system is "the part of the nervous system consisting of nerves and ganglia outside the brain and spinal cord." *Dorland's* 1832.
[5] Dysphagia is "difficulty in swallowing." *Dorland's* 573.
[6] Benign prostatic hyperplasia is an "age-associated enlargement of the prostate resulting from proliferation of both glandular and stromal elements, beginning generally in fifth decade of life; it may cause urethral compression and obstruction." *Dorland's* 882.
[7] Pet. Ex. 12 at 26-30, 223-26.
[8] An intramuscular lipoma is a "slow-growing infiltrating lesion composed of mature fat cells" occurring within the muscle. *Dorland's* 1049.
[9] Pet. Ex. 12 at 32-35, 95-98, 167-220.
[10] *Id.* at 239-268.
[11] *Id.* at 41-68.

and transient cerebral ischemia;[12] a mass in his neck;[13] spondylosis of the cervical spine, [14] lower urinary tract symptoms, and depression.[15]

In 2010, petitioner received treatment for numbness in his hand;[16] memory disorder and dementia;[17] depression, urinary tract retention, hypertension, vitamin D deficiency, anemia, and dysphagia;[18] gastrointestinal issues, diverticulosis,[19] iron deficiency, and esophagitis;[20] mental health issues resulting in unemployment;[21] and chronic low back pain.[22] Petitioner received flu and Adacel[23] vaccines without event. Pet. Ex. 4 at 90-91, 94.

In 2011, petitioner was treated for hypertension, diverticulosis, lipoma, syncope, chronic lower back pain, cervical spondylosis, urinary tract symptoms with trouble holding urine, Vitamin D deficiency, depression, and uncontrolled Type 2 diabetes while taking Glipizide.[24] Pet. Ex. 8 at 5-14. He was provided with blood glucose testing materials. Pet. Ex. 4 at 113, 119-122; Pet. Ex. 94 at 7, 40, 42. His medical records show he was unemployed and was seeking workers' compensation at that time. Pet. Ex. 4 at 113-12; Pet. Ex. 94 at 52, 54.

No records were filed from September 2011 until June 2012, when petitioner contacted Kaiser for a refill of Hydrocodone-Acetaminophen[25] and Simvastatin,[26] a statin. Pet. Ex. 94 at 59, 64. He was advised that his diabetes medications could not be refilled unless labs were done, even if he no longer had insurance. Pet. Ex. 94 at 70. CVS Pharmacy records document refills

[12] Pet. Ex. 12 at 103-09.
[13] *Id*. at 79-84.
[14] Cervical spondylosis is a "degenerative joint disease affecting the cervical vertebrae, intervertebral disks, and surrounding ligaments and connective tissues, sometimes with pain or paresthesia radiating along the upper limbs as a result of pressure on the nerve roots." *Dorland's* 1725.
[15] Pet. Ex. 12 at 227-232.
[16] *Id*. at 271-79.
[17] *Id*. at 280-91.
[18] Pet. Ex. 4 at 27-33; Pet. Ex. 12 at 295-302.
[19] Diverticulosis is the "presence of diverticula, particularly of colonic diverticula, in the absence of inflammation." *Dorland's* 552. Diverticula are "a circumscribed pouch or sac of variable size occurring normally or created by herniation of the lining mucous membrane through a defect in the muscular coat of a tubular organ." *Dorland's* 552.
[20] Pet. Ex. 4 at 43-48; Pet. Ex.12 at 304-311.
[21] Pet. Ex. 4 at 58-67; 97-101.
[22] *Id*. at 68-72.
[23] Adacel is a tetanus toxoid, reduced diphtheria toxoid, and acellular pertussis (Tdap) vaccine. *Dorland's* 25.
[24] Glipizide is an orally administered sulfonylurea compound used as a hypoglycemic in the treatment of Type 2 diabetes mellitus. *Dorland's* 776.
[25] Hydrocodone is "a semisynthetic opioid analgesic derived from codeine but having more powerful sedative and analgesic effects." *Dorland's* 866. Acetaminophen is "the amide of acetic acid and *p*-aminophenol, having analgesic and antipyretic effects similar to those of aspirin but only weak anti-inflammatory effects." *Dorland's* 11.
[26] Simvastatin is "an antihyperlipidemic agent that is an HMG-CoA reductase inhibitor, used to lower blood lipid levels in the treatment of hypercholesterolemia and other forms of dyslipidemia." *Dorland's* 1690.

throughout 2012 for Simvastatin, Hydrocodone,[27] Atenolol,[28] Mirtazapine,[29] Vicodin,[30] and Remeron.[31] Pet. Ex. 92 at 1-2; Pet. Ex. 94 at 101, 104, 109. He received a flu vaccine on October 10, 2012 without event. Pet. Ex. 92 at 2.

CVS Pharmacy records from 2013 reflect refills of Simvastatin, Atenolol, Hydrocodone, Mirtazapine, Lisinopril,[32] and Metformin[33]. Petitioner did not refill any medications to treat his diabetes between December 29, 2012 and March 2013. Pet. Ex. 93 at 1-2.

On March 20, 2013, petitioner presented to Dr. Hopkins to establish care. The record documented uncontrolled Type 2 DM, peripheral vascular complication, urinary incontinence, chronic bac and hip pain, and GERD. Pet. Ex. 9 at 1. Dr. Hopkins noted a need for medication change to control his comorbidities. *Id*. at 2-5.

Petitioner returned to Dr. Hopkins on April 24, 2013. The record documented "poorly controlled DM and bilateral foot pain with neuropathy for which he limps around." Pet. Ex. 9 at 6. He had anemia; gastritis[34] with erosion of the upper GI tract treated with Prilosec and likely slow blood loss; uncontrolled Type 2 diabetes with peripheral vascular complication that was stable with neuropathy treated with Gabapentin; chronic lower back pain; hip pain; and arthritis. *Id*. at 10.

2. Petitioner's Health After Receiving the Influenza Vaccine

On August 19, 2013, petitioner received the subject flu vaccine at CVS Pharmacy. Pet. Ex. 93 at 2.

a. 2013

Petitioner presented to Dr. Hopkins on September 17, 2013, reporting body pain all over after a flu vaccine. "He woke up with right leg pain and weakness. He has had poorly controlled diabetes but now has joint pain and stiffness and has difficulty opening and closing his hand. He denies a reaction prior to this of a rash or other reaction. His blood sugars are stable at home. The

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[27] Hydrocodone is "a semisynthetic opioid analgesic derived from codeine but having a more powerful sedative and analgesic effect." *Dorland's* 867.

[28] Atenolol is "a cardioselective β_1-adrenergic blocking agent used in the treatment of hypertension and chronic angina pectoris and the prophylaxis and treatment of myocardial infarction and cardiac arrythmias." *Dorland's* 169.

[29] Mirtazapine is "an antidepressant compound unrelated to any of the classes of antidepressants; administered orally." *Dorland's* 1152.

[30] Vicodin is a "trademark for combination preparation of hydrocodone bitartrate and acetaminophen." *Dorland's* 2025. Hydrocodone bitartrate is "the bitartrate salt of hydrocodone, used as an analgesic and antitussive, administered orally." *Dorland's* 867. Acetaminophen is "the amide of acetic acid and *p*-aminophenol, having analgesic and antipyretic effects similar to those of aspirin but only weak anti-inflammatory effects." *Dorland's* 11.

[31] Remeron is a "trademark for a preparation of mirtazapine." *Dorland's* 1597. Mirtazapine is "an antidepressant compound unrelated to any of the classes of antidepressants; administered orally." *Dorland's* 1152.

[32] Lisinopril is "the lysine derivative of the active form of enalapril; an angiotensin-converting enzyme inhibitor used in the treatment of hypertension . . . congestive heart failure, and acute myocardial infarction." *Dorland's* 1052.

[33] Metformin is "a biguanide antihyperglycemic agent that potentiates the action of insulin, used in the treatment of type 2 diabetes mellitus; administered orally." *Dorland's* 1129.

[34] Gastritis is "inflammation of the stomach." *Dorland's* 754.

flu shot was given two weeks" ago. Pet. Ex. 9 at 11. He tried ice and heat with some improvement and used Vicodin and Tramadol. Pet. Ex. 9 at 11; Pet. Ex. 2 at 1. An examination revealed global joint stiffness and pain with slow movements, but no inflammatory synovitis or rash. Pet. Ex. 9 at 12; Pet. Ex. 2 at 2. The assessment was acute arthritis and concern for flu vaccine reaction. Petitioner was prescribed a steroid dose pack and Vicodin and advised to report the reaction to CVS. Blood work for erythrocyte sedimentation rate ("ESR")[35] and complete blood count ("CBC") was normal. Pet. Ex. 9 at 13; Pet. Ex. 2 at 3.

Twelve days later, on September 29, 2013, petitioner presented to the emergency room at Sutter Roseville Medical Center and came under the care of Dr. Tandon. Petitioner reported debilitating joint pain throughout his body with generalized aches and pains starting a day after he received a flu shot on August 19, 2013. Pet. Ex. 3 at 2. He did not feel well for two or three days after the flu shot and was unable to go to work. His symptoms progressed and worsened gradually over the next several weeks, causing difficulty getting out of bed, up from a chair, or off the toilet, and he had stiff joints and achy muscles in both the upper and lower extremities, mostly in the shoulder and the thigh. He tried over-the-counter medication, heat, and ice but did not feel better. *Id*. at 8, 22-23. He had some relief from prednisone but has severe pain and muscle weakness throughout his body. Onset was six weeks ago that worsened and is now severe. *Id*. at 2. He had no loss of appetite, headache, visual disturbance, or decreased urine output. *Id*. at 2, 4, 8-9, 24. On examination, he had generalized muscle pains and discomfort, especially in the proximal muscles around the shoulders and thighs with no synovitis, joint swelling, or effusion. There were no focal sensory or motor deficits and cranial nerves II-XII were intact. The assessment was diffuse arthralgias, myalgias, and diffuse generalized weakness, with a history of Type 2 DM, hypertension, and multiple other problems currently stable. *Id*. at 10. Dr. Tandon wrote, "It is possible that he may have had an adverse reaction to the flu shot as his symptoms came after the flu shot was given, and at this time, I will put flu shot in his allergy list." Dr. Tandon noted significant resolution of symptoms with steroids, so Solu-Medrol was ordered, and a neurological consult was ordered due to his neuropathy. *Id*. at 10, 22-24.

Petitioner was examined by a neurologist, Dr. Mahmood and reported onset of symptoms including generalized aches, particularly in the lower extremities, the day after a flu vaccine. By the end of the day or the next day, his symptoms were progressively worse, and it was difficult for him to walk. His right shoulder was first affected, then over the next several days both shoulders had pain, tenderness, and weakness. Steroids worked, but symptoms returned and worsened within a few days of completion. That morning, he had problems getting out of bed and came to the emergency room. Pet. Ex. 3 at 26. Dr. Mahmood noted significant grip strength weakness, significant proximal muscle weakness in both upper and lower extremities, trace reflexes in his knees and upper extremities except for the triceps, and no reflexes in his ankles. Toes were equivocal, there was no significant dystaxia,[36] and sensory exam was normal. The impression was generalized muscle weakness, arthralgias, and myalgias with a history of hypertension, DM, and traumatic spine injury. He believed petitioner's symptoms were related to the flu vaccine and

[35] Erythrocyte sedimentation rate ("ESR") is a non-specific test used to detect illness associated with acute and chronic infection, inflammation, and tissue necrosis or infarction. *Mosby's Manual of Diagnostic and Laboratory Tests* 199 (Pagana eds., 6th ed. 2018) [hereinafter Mosby's].

[36] Dystaxia is "difficulty in controlling voluntary movements." *Dorland's* 576.

suggestive of GBS, but GBS "would not explain the muscle tenderness or soreness." IVIG was recommended with occasional monitoring of his breathing. *Id*. at 27-28.

Petitioner was also examined by Dr. Rangi for chronic anemia. He reported GERD and intermittent but worsening difficulty with swallowing over the "past several months." Pet. Ex. 3 at 29. The assessment was microcytic iron deficiency and chronic anemia, GERD, and dysphagia. Additional outpatient testing was recommended. *Id*. at 30. A CT scan showed a subtle esophageal stricture. *Id*. at 35-36.

Petitioner was discharged on October 4, 2013 with a diagnosis of GBS as a complication of flu shot, esophageal stricture, large hiatal hernia, Cameron ulcer[37] with gastric ulcers, anemia, iron deficiency probably secondary to gastrointestinal bleed, hypertension, DM, and elevated C-reactive protein ("CRP")[38] and ESR. Pet. Ex. 3 at 37.

Petitioner returned to the ER on October 29, 2013, with complaints of muscle and joint aches, weakness, and difficulty walking. He had a history of "atypical GBS (associated with myalgias/arthralgias)" following a flu vaccine. Blood testing was negative for aldolase, rheumatoid arthritis ("RA") quantitative,[39] and antinuclear antibody ("ANA").[40] CRP and ESR were elevated but trended down with IVIG and prednisone. Pet. Ex. 3 at 60. Examination demonstrated trace but symmetric bilateral patellar reflexes, 4+ to 5/5 strength throughout all upper and lower extremities, and 3+ to 4/5 strength on upper abduction of the shoulders and bilateral thigh flexion. Sensory was intact to light touch. The assessment was proximal muscle weakness, myalgias, arthralgias, "possible neuromuscular disorder of unknown etiology at this point," DM Type 2, hypertension, and iron deficiency anemia. *Id*. at 62. Petitioner was admitted and a lumbar puncture was ordered. An MRI could not be done due to the implanted stimulator in his back. *Id*. at 63. A CT scan showed moderate osteoarthritic degenerative changes of both hips, mild tricompartmental osteoarthritic degenerative changes of the knees with small right and trace left joint effusion, colonic diverticulosis, and post-surgical changes of the lumbar spine and iliac bones. *Id*. at 73. His head CT was negative. *Id*. at 74.

Dr. Mahmood examined petitioner upon admission. Petitioner reported he was "convinced he was completely better" when discharged three weeks ago, but then noted worsening weakness and painful movement that progressed until two days ago, when it significantly worsened. Pet. Ex. 3 at 69. On examination, he could move all four extremities but had significant muscle weakness and pain, as well as tenderness and soreness involving the proximal and some distal muscle groups. Tone was largely flaccid with deep tendon reflexes barely elicitable, and toes silent bilaterally. Sensations were intact with equal and full light touch and temperature sensation in both upper and

[37] A Cameron ulcer is "a peptic ulcer with a sliding hiatal hernia; it may be accompanied by chronic bleeding or be clinically silent." *Dorland's* 1967.

[38] C-reactive protein ("CRP") is a protein used to indicate an inflammatory illness. It is elevated in patients with a bacterial infectious disease, tissue necrosis, or an inflammatory disorder. A positive test result indicates the presence, but not the cause, of the disease. *Mosby's* 165-66").

[39] Rheumatoid arthritis ("RA") quantitative, or the RA Factor, is a test used in the diagnosis of RA that is directed toward identification of the IgM antibodies. *Mosby's* 409-410.

[40] Antinuclear antibodies ("ANA") are used to diagnose systemic lupus erythematosus and other autoimmune diseases, including but not limited to RA, polymyositis, scleroderma, infectious mononucleosis, and myasthenia gravis. *Mosby's* 80-83.

lower extremities. *Id*. at 70. The impression was polymyositis and possible paresthesia, with a history of DM, hypertension, and lower spine disease. *Id*. at 70-71. Dr. Mahmood recommended 48-72 hours of steroids. *Id*. at 71.

A rheumatologist, Dr. Bhat was consulted on October 30, 2013 to rule out inflammatory polymyositis. Pet. Ex. 3 at 82. A CT scan of the lower extremities and CK and aldolase levels were normal, so inflammatory polymyositis was less likely, and he was not toxic appearing, so it was not serum sickness. Aseptic meningitis[41] was added to the differential diagnosis. *Id*.

Petitioner was discharged on October 31, 2013 with a diagnosis of steroid-responsive proximal myopathy[42] of unclear etiology, recent history of GBS probably related to flu vaccine, abnormal lumbar puncture with aseptic meningitis without typical meningitis symptoms, DM, implanted stimulator device, and probable allergy/reaction to flu vaccine. Pet. Ex. 3 at 88.

Petitioner returned to Dr. Hopkins on November 12, 2013 and reported feeling weak and tired, with decreased strength in his legs and hands, paresthesia, and tingling in his hands, but he felt better on steroids. He took narcotics for chronic back pain. A muscle biopsy was being entertained. Pet. Ex. 2 at 4. On examination, there was no edema, 4/5 strength in the lower extremities, 3/5 grip strength, and no ataxia with fair balance. *Id*. at 5. Dr. Hopkins noted GBS, a flu shot, two hospitalizations, numerous neurology and rheumatology consults, petitioner feeling tired and lethargic with recurrent symptoms when steroids were decreased, and diabetic neuropathy with neurologic and peripheral vascular complication that "is not part of his above syndrome." He needed to be on disability for at least 90 days and have assistance at home. *Id*. at 9-10. Petitioner filed for disability on December 5, 2013 for Guillain-Barré Syndrome. Pet. Ex. 108 at 2, 239.

On December 9, 2013, petitioner presented to Dr. Seminer for outpatient neurological care of acute weakness and "possible GBS." Pet. Ex. 3 at 97. Petitioner reported onset of symptoms the day after the flu vaccine with gradual weakness over the next several weeks. He was treated with IV Solu-Medrol, improved, and was diagnosed with "steroid-responsive myopathy." He had not had any steroids for two weeks and his current complaints included pain in all joints, occasional numbness and tingling in his right foot, and inability to open a jar. He did not have dysarthria,[43] diplopia,[44] dysphagia, or eyelid ptosis,[45] and there was no muscle atrophy[46] or fasciculations.[47] *Id*. at 97-98. Petitioner reported a four-month history of relapsing muscle weakness with some sensory complaints. His CPK and sedimentation rate were normal, therefore myositis was unlikely. He may have developed demyelinating neuropathy following the flu vaccine, but current findings

[41] Aseptic meningitis refers to "any of several types of mild meningitis, most of which are caused by viruses." *Dorland's* 1117. Meningitis is inflammation of the meninges. *Dorland's* 1117.
[42] Myopathy is defined as "any disease of a muscle." *Dorland's* 1206.
[43] Dysarthria is "a speech disorder consisting of imperfect articulation due to loss of muscular control after damage to the central or peripheral nervous system." *Dorland's* 569.
[44] Diplopia is "the perception of two images of a singular object. *Dorland's* 518.
[45] Ptosis is "drooping of the upper eyelid." *Dorland's* 1527.
[46] Muscle atrophy is "a wasting of muscle tissue." *Dorland's* 173.
[47] Fasciculation is "a small local contraction of muscles, visible through the skin, representing a spontaneous discharge of a number of fibers innervated by a single motor nerve filament." *Dorland's* 675.

suggested CIDP. EMG/NCS testing of the upper and lower extremities was ordered along with an 80 mg taper of prednisone, a follow up on sugar levels, and repeat ESR and CPK testing. *Id*. at 99.

The next day, December 10, 2013, petitioner presented to Dr. Hopkins with complaints of weakness, fatigue, joint pain, and leg and hand numbness as lingering effects of GBS. His pulse, blood pressure, and blood sugars were elevated by cyclical doses of prednisone. He remained out of work. Pet. Ex. 2 at 13. He was globally weak but non-focal, with no ataxia[48] and slow but steady gait. Sensation was intact and motor strength was 4/5. *Id*. at 14. He was making slow progress, with symptom flares when coming off prednisone. His diabetic neuropathy was noted, and his blood pressure medication was increased. *Id*. at 18.

b. 2014

Petitioner was admitted to Sutter Roseville on January 8, 2014 for weakness likely secondary to steroid response, exacerbation of GBS, and neuropathy of unclear etiology. Pet. Ex. 20 at 1; Pet. Ex. 100 at 1, 3. A lumbar puncture showed no infectious etiology, but elevated protein and glucose levels suggestive of aseptic meningitis. Pet. Ex. 20 at 2; Pet. Ex. 100 at 11. Dr. Mahmood documented a discussion with Drs. Agaiby and Saeed about the combination of symptoms being suggestive of "some kind of neuropathic involvement which did not follow any significant dermatomal pattern." Pet. Ex. 100 at 12. The lack of deep tendon reflexes and history of association with the flu vaccine suggested the possibility of GBS, which, if true, had become chronic and should be considered CIDP. However, petitioner's muscle stiffness, tenderness, and soreness were more suggestive of myositis or myopathy, and the diagnosis was clouded by impressive steroid responsiveness. Dr. Mahmood documented continued concern about possible spinal cord involvement and noted that radiology refused to do an MRI due to petitioner's implanted stimulator. The diagnosis remained elusive; the steroid responsiveness and mixed symptoms with associated urinary incontinence made spinal cord imaging and MRI very important. *Id*. at 12-13.

Petitioner was discharged on January 10, 2014 with weakness secondary to steroid responses, proximal myopathy of unclear etiology, history of GBS complication from flu vaccine, microcytic anemia,[49] and gastritis or gastric ulcer. Pet. Ex. 100 at 8-10.

Petitioner returned to Dr. Hopkins on January 23, 2014. "He has DM and neuropathy" and is on insulin and steroids. He had chronic back and hip pain, and worsening bladder problems with GBS. He had increased urinary symptoms. Pet. Ex. 9 at 14. He looked pale but had no edema; his motor strength was 5/5 with grip; lower extremities were 4/5 with right foot drag, unsteady gait and balance. *Id*. at 15. Dr. Hopkins's assessment was that he may have a GBS variant in addition to diabetic neuropathy with neurological complications. His diabetes was uncontrolled with peripheral vascular complications. *Id*. at 18.

[48] Ataxia is the "failure of muscular coordination; irregularity of muscular action." *Dorland's* 168.

[49] Microcytic anemia is "any anemia characterized by microcytes (erythrocytes that are smaller than normal). *Dorland's* 78. Anemia is "a reduction below normal in the concentration of erythrocytes or hemoglobin in the blood . . . it occurs when the equilibrium is disturbed between blood loss (through bleeding or destruction) and blood production." *Dorland's* 77.

On January 24, 2014, petitioner returned to Dr. Seminer reporting that his hands felt a bit heavy. He had finished prednisone four days ago, was walking with a cane, and was slightly unsteady with right foot drag. He was treated with IV methylprednisolone and a tapering dose of prednisone as an outpatient. He had mild difficulty swallowing. Pet. Ex. 3 at 104. His motor tone was normal, strength was 5+, deep tendon reflexes were absent throughout, and he could support his weight and walk on his heels and toes. He was released to work five hours a day, standing no more than an hour at a time. A muscle biopsy was again considered. Pet. Ex. 3 at 105.

Petitioner had his first electromyography and nerve conduction study (EMG/NCS) on January 24, 2014, which showed a few mild neuropathic electrophysiological abnormalities with mild prolongation of sensory and motor nerve conduction across the wrist and right median nerve consistent with right sided carpal tunnel syndrome. There was prolongation of F-wave latencies in the right ulnar[50] and tibial nerves[51], which were isolated findings of "questionable" significance. There was no other evidence of conduction slowing or neuropathy in the upper and lower extremities. It was concluded that, "[T]he findings do not support a clinical diagnosis of Guillain-Barre syndrome or CIDP." Pet. Ex. 3 at 106-07.

A psychological examination conducted by the Department of Social Services on February 12, 2014 noted normal gross motor function and able to ambulate without assistance. He reported depression about his inability to find treatment for his condition that started in October 2013 and has worsened since. He was stressed about being able to care for himself. Pet. Ex. 108 at 142-43. Petitioner reported a steady work history; though not currently working, he was employed by West Marine and had worked there for 15 years as a manager.[52] He reported no problems with managers or coworkers in the past. *Id*.[53] He was independent in activities of daily living and reportedly spent his days eating, sleeping, and watching TV. *Id*. at 144. Mental health services for his depression were suggested. He was determined not to be disabled and was denied Social Security Disability Benefits. Pet. Ex. 108 at 145-46, 148-49.

Petitioner returned to Dr. Seminer on February 24, 2014, complaining of worsening weakness in the upper and lower extremities, slight difficulty swallowing, and frequent urination. Pet. Ex. 3 at 112. Dr. Seminer wrote, "Based on patient's symptoms and slightly abnormal electrodiagnostic studies, I think that he probably have (sic) CIDP." He ordered IVIG three times per month. *Id.* at 113.

On March 24, 2014, Petitioner returned to Dr. Hopkins for a check-up, was noted to be in respiratory distress with chest tightness and was transported to the hospital by ambulance and admitted. Pet. Ex. 2 at 20, 24. He was discharged on March 27, 2014 with shortness of breath secondary to probable CIDP; history of GBS; hypertension; Type 2 DM made worse by steroids; history of gastric ulcer; probable thalassemia;[54] and resolved acute kidney injury. Pet. Ex. 3 at 117.

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[50] The ulnar nerve is the nerve "pertaining to the ulna or to the medial aspect of the forearm as compared with the lateral (radial) aspect." *Dorland's* 1968.

[51] The tibial nerve is the nerve "pertaining to the tibia or to the medial aspect of the leg." *Dorland's* 1897.

[52] This is inaccurate. The evidence shows petitioner last worked for West Marine in 2007. He was rehired in 2012 as an employee, not a manager. Tr. 86-87; Pet. Ex. 101 at 22, 29-31, 41, 113.

[53] Petitioner's records document an incident with a coworker in 2010. Pet. Ex. 4 at 59.

[54] Thalassemia is "a heterogenous group of hereditary hemolytic anemias that have in common a decreased rate of synthesis of one or more hemoglobin polypeptide chains and are classified according to the chain involved." *Dorland's*

At his April 21, 2014 visit, Dr. Seminer noted that the EMG/NCS testing done on March 26, 2014 revealed no real prolongation or absence of F-waves in the lower extremities, with an incidental finding of median neuropathy at the right wrist. The test was otherwise normal, as was the repetitive stimulation testing performed. Monthly IVIG infusions were ordered for CIDP and work accommodations of no climbing or heavy lifting recommended. Pet. Ex. 3 at 152.[55]

On April 28, 2014, petitioner filed for reconsideration of his Social Security disability denial submitting other unspecified arthropathies and diabetes mellitus, with a date of disability of October 29, 2013. Pet. Ex. 108 at 239. He was determined to be disabled as of April 2014, with payments starting June 2014 with back payments approved for April and May. *Id*. at 240-41, 245.[56]

On May 9, 2014, petitioner presented to Dr. Knox advising that his symptoms worsened two weeks after the March IVIG infusion and within four weeks he had difficulty moving his legs. On the date of this visit, he was on the third day of a three-day course of IVIG and was doing better. Pet. Ex. 5 at 4. Examination revealed normal strength, tone, sensation, and gait. Dr. Knox's impression was "possible CIDP" given the clinical response to IVIG and marked proximal weakness. IVIG treatments were to continue, tapered every two weeks. Pet. Ex. 5 at 4, 6, 16. IVIG was necessary due to risks from prednisone with petitioner's history of stroke and DM. *Id*. at 6.

Genetic testing conducted on May 9, 2014 resulted in Dr. Knox documenting "CANNOT BE ON IMURAN", which petitioner had taken in the past. Pet. Ex. 5 at 21; Pet. Ex. 89 at 48.

Petitioner returned to Dr. Knox on June 11, 2014. His last infusion was June 6, 2014. He reported he felt good until Sunday, when he awoke tired with difficulty getting his legs to move and fell. His legs felt like they were dragging. Pet. Ex. 5 at 18.

Petitioner presented to Dr. Hopkins on June 19, 2014 reporting exacerbation of CIDP. He was using a cane and complained of fatigue, weakness, falling, and feeling depressed. Pet. Ex. 2 at 25; Pet. Ex. 9 at 19. He appeared tired on examination, with symmetrical weakness and unsteady gait but without tremor or ataxia. Pet. Ex. 2 at 26; Pet. Ex. 9 at 20. The assessment was CIDP. Petitioner was directed to restart steroids, Levemir for diabetes, and continue his other medications. His hypertension was stable; CIDP was the primary issue. Pet. Ex. 2 at 30.

On July 9, 2014, petitioner returned to Dr. Knox, who noted that while intravenous Solu-Medrol (IVSM) worked better than IVIG, petitioner's blood sugar was uncontrolled on it. Pet. Ex. 5 at 34. Petitioner followed up with Dr. Knox again on July 17, 2014. *Id*. at 40. He had no clear weakness but had slight foot drop with walking. He was released to work 20 hours per week. *Id*. at 43. Petitioner received IVIG infusions through August 1, 2014. Pet. Ex. 3 at 154-219.

1878. Hemolytic anemias are "any of a group of acute or chronic anemias characterized by excessive hemolysis and impaired erythropoiesis." *Dorland's* 78.

[55] It is unclear from the records whether petitioner was working at this time since he had been awarded disability.

[56] Petitioner was ultimately awarded Medicare Part A (hospital insurance) and Part B (medical insurance) beginning in April 2016. Pet. Ex. 108 at 248; Pet. Ex. 109.

On August 25, 2014, at an appointment with Dr. Hopkins, petitioner reported working 20 hours a week. His weakness was stable. He was taking Levemir,[57] Metformin, Cellcept,[58] and Solu-Medrol, which affected his DM. He had fatigue, lethargy, GI upset, and weakness. Pet. Ex. 2 at 32; Pet. Ex. 9 at 26. Dr. Hopkins's assessment was CIDP; chronic but stable back pain; diabetic neuropathy with neurologic complication, stable with mixed presentation given his primary neurologic disorder; hypertension; and diabetes. Pet. Ex. 2 at 40; Pet. Ex. 9 at 34.

Petitioner returned to Dr. Knox on September 9, 2014. He was receiving IVIG infusions on Mondays and Thursdays but felt weak by the weekend. Pet. Ex. 5 at 55. He wanted IVSM three times per week because he began feeling weakness after three days. Pet. Ex. 13 at 296. He saw Dr. Knox again on November 3, 2014. He was receiving infusions three times per week and felt "great and functional." He was stronger but still using a cane. His blood sugar was high. *Id*. at 306, 309. Petitioner returned to Dr. Knox on November 18, 2014, still feeling great with his strength back to normal. Blood sugar level was still high. *Id*. at 311.

Another EMG/NCS study was performed on November 18, 2014 and was no different when compared to the March 26, 2014 study. Pet. Ex. 13 at 198-201, 315; Pet. Ex. 3 at 104-08. Dr. Knox wrote "interesting that the ncs never showed demyelinating ????" Pet. Ex. 13 at 315. Petitioner was on full disability at this time. *Id*. at 316.

On November 25, 2014, petitioner was sent by the infusion center to the ER for two days of neck swelling and difficulty swallowing. Pet. Ex. 13 at 345. A small hiatal hernia and mild multilevel cervical spondylosis were noted. *Id*. at 349.

After an examination on December 11, 2014, Dr. Knox documented that petitioner was using a cane but had no power loss. Pet. Ex. 13 at 320. His blood sugar level was 500 and unsustainable. *Id*. at 321.

c. 2015

On January 13, 2015, Dr. Knox noted a recent ER visit for cellulitis of the left leg on December 27, 2014. Petitioner needed help to stand, had slight weakness, and no reflexes. He was directed to secure and see a new PCP before February since Dr. Hopkins no longer accepted his insurance. Pet. Ex. 13 at 321-26.

At a visit to Dr. Knox on February 12, 2015, petitioner complained of weakness. He was unable to get subcutaneous methotrexate, so he was using pills and taking folic acid. Petitioner reported his blood sugar was less than 200 now that he was off steroids. He claimed to be getting weaker, falling, and having difficulty chewing. Pet. Ex. 20 at 5. Dr. Knox again advised he needed a PCP for diabetes control. *Id*. at 5, 10.

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[57] Levemir is a "trademark for a preparation of insulin detemir." *Dorland's* 1018. Insulin detemir is "a long-acting insulin analogue in which the threonine at position 30 of the B insulin chain is omitted, and a 14-carbon fatty acid chain is attached to the amino acid at position 29; administered subcutaneously in the treatment of diabetes mellitus." *Dorland's* 934.

[58] Cellcept is a "trademark for preparations of mycophenolate mofetil." *Dorland's* 320. Mycophenolate mofetil is an immunosuppressive agent. *Dorland's* 1199.

On February 16, 2015, petitioner presented to a new PCP, Dr. Belsky. Pet. Ex. 20 at 10-12. Dr. Belsky's assessment was uncontrolled Type 2 DM with peripheral vascular complication; hypertension well-controlled on Lisinopril and Atenolol; high cholesterol controlled on statins; history of transient ischemic attack and stroke; CIDP followed by neurology; and anemia followed by hematology. He also had suspected thalassemia for which he received IV iron infusion. *Id*. at 14.[59] On March 13, 2015, Dr. Belsky documented that Medi-Cal no longer covered freelance glucometers and petitioner had not tested his sugar since he ran out of strips on March 6, 2015. *Id*. at 15. On March 16, 2015, petitioner presented for diabetes education and insulin management. Steroid use elevated his blood sugar to the 500s. *Id*. at 18-19.

On March 18, 2015, petitioner returned to Dr. Knox, who documented that petitioner was self-prescribing 20 mg of prednisone four times per day for the past month and a half. Pet. Ex. 20 at 19. The impression was still CIDP, though the clinical diagnosis was "concerning." He was to resume IV steroids at 1 mg once per week. Pet. Ex. 20 at 25.

On March 20, 2015, petitioner returned to Dr. Belsky frustrated by the uncertainty of his diagnosis. Dr. Belsky ordered IV steroid infusions but cautioned petitioner against taking the 80 mg of oral steroids daily that he was self-prescribing. Pet. Ex. 20 at 26.

On May 1, 2015, petitioner saw Dr. Ambriz for an eye examination and was cautioned to control his blood sugar to avoid diabetic eye complications and the risk of blindness. *Id*. at 31.

On May 20, 2015, petitioner returned to Dr. Knox with difficulty swallowing and choking on food, losing his voice, and water coming through his nose and mouth when drinking. He continued to self-prescribe 80 mg of prednisone per day. Pet. Ex. 20 at 32.

At the time of his June 24, 2015 visit with Dr. Knox, petitioner had received IVIG/Solu-Medrol, felt great with "some power back," and could stand, walk, talk, and breathe. Dr. Knox wrote, "The question has always been the diagnosis…" An examination showed "slight weakness infraspinatus. Slight weakness of hip flexion. The rest of the power is strong." Pet. Ex. 20 at 46. A NCS of the right upper and lower limb on that date were comparable to those performed on March 26, 2014. Repetitive stimulation testing showed no decremental responses. The impression was "possible primary muscle disorder. Normal CPK argues against a muscle disorder affecting the muscles themselves and thus I suspect we may be dealing with an inflammatory response muscle disorder such as seen in sarcoid." Dr. Knox noted a need for a muscle biopsy, a second opinion, a referral to neurosurgery, and continuing intervention with steroids, with more directed therapies "once we have a better sense of what is going on." *Id*. at 47. In a July 1, 2015 addendum, Dr. Knox wrote that a muscle biopsy should be done on the left. Petitioner cannot have an MRI because he claims the spinal stimulator cannot be removed. *Id*. at 48.

At his July 17, 2015 visit, Dr. Knox noted petitioner had an infusion three days prior and was feeling better but still had problems swallowing at times. A muscle biopsy was scheduled for August 3, 2015.[60] Pet. Ex. 20 at 48. He saw Dr. Belsky for a right wrist injury from a fall five days later and reported struggling with lower leg weakness. Pet. Ex. 16 at 2.

[59] No records associated with iron infusions were filed.
[60] The scheduled muscle biopsy was ultimately cancelled due to loss of insurance. Pet. Ex. 18 at 1.

Petitioner was examined by Dr. Carroll on August 26, 2015 for microcytic anemia with iron deficiency that did not respond to iron supplementation, due to malabsorption and alpha thalassemia trait. He was to be treated with intravenous iron Dextran quarterly as needed to keep his ferritin greater than 100. Pet. Ex. 20 at 61.

Petitioner returned to Dr. Knox on October 1, 2015 and reported feeling better, swallowing better, walking great, and being better able to stand up from a chair, with no heaviness and barely any numbness in his hands. Pet. Ex. 18 at 1. Dr. Knox wrote, "Possible myopathy but with numbness places this into the cidp or similar Could be myelopathy but has no spasticity Responsive to steroids Not (sic) sign abnormal emg Needs muscle biopsy The muscle biopsy should be done on the left, repeat the left iliopsoas." *Id*. at 7.

Petitioner was admitted to the hospital overnight on December 8, 2015 for chest pain, shortness of breath, and possible loss of consciousness during an IVIG infusion. Pet. Ex. 20 at 79. Cardiac testing was negative. *Id*. at 197, 206-07.

d. 2016

At a January 26, 2016 visit, Dr. Knox wrote "it would be nice ot (sic) confirm cidp need another ncs and then muscle biopsy." He was okay with continuing IVIG for now. Pet. Ex. 20 at 102. At his March 18, 2016 visit, petitioner reported feeling great and "back to me again." Dr. Knox noted petitioner mixes power with sensory loss when he speaks, which is "hard to separate out." *Id*. at 103.

On March 25, 2016, Dr. Belsky discussed petitioner's elevated creatinine and kidney injury with unclear etiology, possibly related to infusions. Pet. Ex. 20 at 110. On April 1, 2016, Dr. Belsky stopped Metformin and Lisinopril and ordered urinalysis and a renal ultrasound. *Id*. at 116. Dr. Belsky saw him regularly over the next several months for ongoing kidney issues. *See* Pet. Ex. 20.

On August 11, 2016, Medicare denied petitioner's IVIG (Privigen) treatments for failure to meet authorization requirements, which include EMG findings of two of the three criteria: reduced conduction velocity of 2 or more motor nerves; prolonged distal latency of 2 or more motor nerves; prolonged F-wave latencies of 2 or more motor nerves or the absence of F-waves. Pet. Ex. 24 at 1. An appeal was filed and on October 7, 2016, petitioner's Privigen was approved from August 10, 2016 through January 7, 2017. Pet. Ex. 24 at 20; Pet. Ex. 98 at 2.

At an August 18, 2016 visit, petitioner reported feeling great receiving weekly IVIG infusions and prednisone. Pet. Ex. 20 at 147. Dr. Knox's impression was lower extremity weakness responsive to IVIG and prednisone and "[p]robable CIDP," but the only supportive evidence was prolonged F-waves/H reflexes. Another lumbar puncture "was not worth it." *Id*. at 148. He prescribed 150 mg of Imuran a day with hopes of weaning him from IV medications. *Id*. Petitioner continued to receive IVIG through the end of 2016. *See* Pet. Ex. 22.

On December 21, 2016, Dr. Belsky wrote a letter on petitioner's behalf stating that he had CIDP and would benefit from an increase to the 16 hours of daily skilled home care he was receiving. Pet. Ex. 23.

e. 2017

On January 30, 2017, Dr. Knox wrote "Inflammatory NM (neuromuscular) disease Likely CIDP Confirmed by clear clinical presene (sic) of response to IVIG But testing limited Cannot get mri Is (sic) spine LP-OK EMG – prolonged F-waves. But with numbness places this into the cidp or similar Maybe - with immune modulators will improve." Pet. Ex. 89 at 9.

On February 3, 2017, Dr. Belsky again advised petitioner about his self-prescribing of 80 mg a day of corticosteroids in addition to what his physicians prescribed. He strongly encouraged petitioner to stop, as it complicated the management of his comorbidities. Pet. Ex. 89 at 11-12. Dr. Belsky noted that Dr. Knox had reduced petitioner's Solu-Medrol dose by half. *Id*. at 11.

On March 27, 2017, petitioner presented to Dr. McMullen for diabetes follow up. He was not tracking his sugars or how much insulin he was taking but advised he took between 20 and 40 units of Humalog[61] before meals, though it was unclear how he decided how much to take. Pet. Ex. 89 at 16-17. He had a caretaker during the day. *Id*. at 17.

By June 30, 2017, petitioner's IVIG infusions had stopped, and his sugar levels were better. He felt his overall strength was stable, but he continued to struggle with walking long distances and used a cane or ski poles to do so. Pet. Ex. 89 at 40.

On July 13, 2017, petitioner presented to the ER with worsening weakness, pain, leg swelling, numbness and a history of CIDP. He reported having no IVIG treatment since May 23, 2017. Pet. Ex. 89 at 218. An ultrasound for deep vein thrombosis was negative. *Id*. at 225. His symptoms were consistent with cellulitis, and he was prescribed Bactrim. *Id*. at 223.

Petitioner returned to the ER on July 28, 2017 with leg redness and swelling after finishing the antibiotics with no relief. He reported receiving IVIG for three years for CIDP which was discontinued 47 days ago. Pet. Ex. 89 at 237. Dr. Lapin noted bilateral lower extremity swelling and erythema with possible elements of cellulitis. Due to his DM, petitioner was prescribed Zosyn[62] and Vancomycin[63] and an echocardiogram was ordered to rule out heart failure. There was no compartment syndrome or compromised renal function. His international normalized ratio ("INR")[64] was 1 with lower extremity bruising, and some capillary fragility secondary to trauma. Neurology was consulted due to progressive weakness since his last treatment in May. *Id*. at 248. He was discharged on August 1, 2017. The discharge summary documented DM and cellulitis of

[61] Humalog is a "trademark for preparations of insulin lispro." *Dorland's* 863. Insulin lispro is "a rapid-acting insulin analogue in which the lysine and proline residues at positions at 28 and 29 on the insulin B chain are reversed; administered subcutaneously in treatment of diabetes mellitus." *Dorland's* 934.

[62] Zosyn is a "trademark for combination preparations of piperacillin sodium and tazobactam sodium." *Dorland's* 2065. Piperacillin sodium is "the sodium salt of piperacillin, used in the treatment of infections caused by susceptible organisms and in intra-abdominal surgery for the prevention of infection; administered intramuscularly or intravenously." *Dorland's* 1428. Tazobactam sodium is "the sodium salt of tazobactam, used in combination with piperacillin sodium to broaden its spectrum of activity against beta-lactamase producing organisms." *Dorland's* 1845.

[63] Vancomycin is "an antibiotic . . . which is highly effective against cocci, especially staphylococci, and other gram-positive bacteria." *Dorland's* 1993.

[64] International normalized ratio ("INR"), also known as prothrombin time ("PT"), is used to evaluate the adequacy of the extrinsic system and common pathway in the clotting mechanism. It measures the clotting ability of factors I (fibrinogen), II (prothrombin), V, VII, and X (ie, the extrinsic system and common pathway). *Mosby's* 391-93.

the left lower extremity. He received one IVIG treatment and was prescribed three days of Keflex for cellulitis, which was nearly resolved on discharge. *Id*. at 275-79.[65]

On August 7, 2017, petitioner presented to Dr. McMullen. He reported he had not received IVIG since May but had been hospitalized for IV antibiotics for cellulitis. His legs were still swollen, and his arms and feet felt heavy and numb. He changed his insulin dose on his own and was not having excessive urination or thirst. Pet. Ex. 89 at 52. Petitioner also presented to Dr. Carroll on the same day for anemia responding well to iron supplementation, but it appeared he had a malabsorption problem and GI bleeding with thalassemia trait. Dr. Carroll advised continued iron dextran[66] supplementation to keep his ferritin greater than 100. The record noted that his CIDP was IVIG, steroid, and Imuran refractory. Rituximab,[67] which was off label, was suggested. *Id*. at 58.

On August 9, 2017 Dr. Knox referred petitioner to Dr. Katz for a second opinion on CIDP and whether Rituximab should be used. Pet. Ex. 89 at 295.

Petitioner presented to Dr. Katz on August 15, 2017 with a four-year history of "CIDP" which started with his legs and arms getting heavier, then his finger started feeling fat and numb. He could not hold himself up, get up and down a ladder, would "trip over himself", and fell several times. His current symptoms included heaviness, neck muscle pain, head drop, tiring easily when walking, truncal weakness, a numb/dumb feeling in his hands, and jolts down his legs which come and go based on his treatment cycle. He received IVIG twice a week for the past two years and said, "he has had two cardiac arrests from IVIG in the past." He has DM and blames his current DM on prednisone. Pet. Ex. 85 at 1; Pet. Ex. 89 at 299. Dr. Katz found no evidence of CIDP, which upset petitioner. Pet. Ex. 85 at 4. Dr. Katz referenced multiple normal NCS studies, the presence of reflexes, and weakness of "breakaway type." *Id*. The CSF protein level on lumbar puncture was explained by IVIG just before testing. Nothing in his history fit GBS and nothing in the chart supported progression. Dr. Katz further noted petitioner has not had any clear recurrence in three months without IVIG and questioned why petitioner could not walk and was in a wheelchair. Pet. Ex. 85 at 4; Pet. Ex. 89 at 302. Dr. Katz ordered another EMG but did not expect to see any difference and deferred to Dr. Knox. Pet. Ex. 85 at 4; Pet. Ex. 89 at 302. Dr. Katz advised that petitioner did not have CIDP. Pet. Ex. 89 at 72. Dr. Knox then changed his impression to inflammatory or pseudo-neurological disease, referred petitioner to rheumatology, and stopped petitioner's medication, which angered petitioner. *Id* at 79-80.

At his October 4, 2017 visit with Dr. Carroll, petitioner expressed anger at Dr. Katz's opinion that he had arthritis rather than CIDP. He had a marked increase in pain and disability since IVIG stopped. Petitioner had not seen the rheumatologist and or been compliant with iron infusions. Pet. Ex. 89 at 83.

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[65] Records from this hospitalization are contained in Pet. Ex. 88 as well.

[66] Iron dextran is "a sterile colloidal solution of ferric hydroxide, $FE(OH)_3$, complexed with partially hydrolyzed low molecular weight dextran, in water for injection; administered intravenously or intramuscularly as a hematinic." *Dorland's* 949.

[67] Rituximab is "a chimeric murine/human monoclonal antibody that binds the CD 20 antigen; used as an antineoplastic in the treatment of CD 20-positive, B-cell non-Hodgkin lymphoma; administered intravenously." *Dorland's* 1624.

Petitioner then presented to the ER on October 14, 2017, reporting a history of CIDP with a "flare-up" of heaviness, weakness, and numbness. His last IVIG infusion was in August because his CIDP diagnosis was questioned by a specialist at UCSF. Pet. Ex. 89 at 316-17. He was admitted for neurological consult with Dr. Byrd, who noted that petitioner's IVIG had been stopped by two neurologists who ruled out CIDP, but petitioner refused to accept this and presented to the ER for treatment. Dr. Byrd refused to order IVIG and suggested physical therapy, but petitioner left against medical advice. *Id*. at 323, 335.

At his visit with Dr. Belsky on October 24, 2017, petitioner said he presented to the ER in hopes of receiving IVIG but left when they would not provide it. He had increasing weakness in his arms and legs over the past two months; his hands felt numb, and he was wearing diapers, so he did not have to get up as much. He used a walker or cane at home but spent most of the day on the couch because he was too tired and weak to walk. He had help during the day. Pet. Ex. 89 at 86.

On October 30, 2017, petitioner saw Dr. Scalapino, a rheumatologist. Pet. Ex. 89 at 87. His assessment was peripheral neuropathic pain with poor balance:

> It is not clear to me whether he has CIDP, CIDP related to diabetes, or some other peripheral neuropathy that involves sensory over motor and is associated with rather severe paresthesias, loss of position and vibratory sensation and distal loss of strength, especially hands. It looks as though his electrophysiologic testing was only lukewarm support for the diagnosis. Against [CIDP] is the fact that his reflexes seem intact but in favor of CIDP is the fact that he has clearly responded temporarily to IVIG and steroids.

Pet. Ex. 89 at 94. He further noted diabetic peripheral neuropathy associated with Type 2 diabetes that is approaching good control, but there is no expectation for the neuropathy to normalize at this point. Absorption problems may be contributing to neurologic symptoms, though it was doubtful given the chronology of problems following vaccination. He was vitamin D deficient with bad degenerative joint disease of the hip contributing to his difficulty getting up and down from a chair, etc., but it does not seem to give him any pain. *Id*. at 94-95.

On November 7, 2017, petitioner presented to Dr. McMullen complaining of numbness and pain in his arms and legs. Dr. McMullen wrote that his diagnosis of CIDP was now questionable; he no longer received infusions but was self-medicating with 20 mg of prednisone three times per day. He declined to have diabetic education sent to his home. Pet. Ex. 89 at 95.

On November 17, 2017, petitioner presented to the ER following a fall that morning injuring his back. He was unable to stand, had a history of leg weakness, hit his head on the wall when he fell, and had numbness through his body all the time. Pet. Ex. 89 at 353. Lower extremity swelling and rash was noted, and he was prescribed Norco[68] for pain. *Id*. at 358.

On December 6, 2017, petitioner returned to Dr. Belsky with right hip pain and acute right-sided low back pain from a fall. Dr. Belsky noted it was likely a contusion, but he was at increased risk for fracture due to ongoing corticosteroid use which he had been warned to stop many times.

[68] Norco is a "trademark for combination preparations of hydrocodone bitartrate and acetaminophen." *Dorland's* 1272.

Pet. Ex. 89 at 100. Petitioner reported spending his days watching TV but enjoys getting out, looking at real estate, and spending time on a friend's boat. He was taking 600 mg of Gabapentin at bedtime. *Id*. at 101. A friend planned to move in to assist him with activities of daily living. *Id*. at 102. He was prescribed Tramadol[69] and referred to orthopedics. *Id*. at 104. X-rays of the right hip showed severe degenerative changes; x-rays of the lumbar spine showed multilevel degenerative changes. *Id*. at 151-52.

Another EMG/NCS on December 20, 2017 showed no changes. Pet. Ex. 89 at 180-84.

f. 2018

On February 13, 2018 petitioner presented to the ER with wrist pain from a fall. Pet. Ex. 89 at 378. X-rays of his wrist, hand, and shoulder showed no fractures and venous doppler of the left extremity showed no deep venous thrombosis. *Id*. at 384, 386.

Petitioner presented to Dr. Latov in New York City on March 8, 2018, complaining of weakness and difficulty walking. Pet. Ex. 58 at 1. He reported generalized weakness three weeks after the flu vaccine in August 2013 for which he was hospitalized and treated with IVIG for CIDP. He reported multiple relapses. IVIG and steroid treatment was discontinued six months ago. He was on Methotrexate in 2015 and Imuran in 2016, both without benefit. His creatinine was elevated, EMG/NCS studies were normal, but he remained weak, using bilateral walking canes. Pet. Ex. 58 at 1-2; Pet. Ex. 63. Another EMG/NCS was ordered.

Petitioner presented to Dr. Belsky for right hip pain in March 2018 and received a cortisone injection. Pet. Ex. 60 at 2. X-rays showed advanced degenerative changes in the right hip, and he underwent hip surgery on May 7, 2018. Pet. Ex. 60 at 6-7, 15; Pet. Ex. 71; Pet. Ex. 59 at 21. During his hospitalization, he was noted to be neurovascularly intact, with limited range of motion of the right leg secondary to right hip surgery. He had normal reflexes. Pet. Ex. 71 at 23.[70]

At a visit with Dr. McMullen on April 6, 2018, Dr. McMullen wrote, "Hx of: neuropathy ? CIPD versus DM." Pet. Ex. 60 at 8.

At a follow up with Dr. Belsky on May 23, 2018, it was noted that petitioner was in a rehabilitation facility, working with a physical therapist three times per week, and taking Percocet. Pet. Ex. 60 at 26. He reported seeing Dr. Latov. Pet. Ex. 60 at 26-27; Pet. Ex. 64.

The EMG/NCS study ordered by Dr. Latov performed on June 21, 2018 was read as abnormal with evidence of chronic generalized sensorimotor polyneuropathy with demyelinating features. Median neuropathy of the right carpal tunnel was noted, most likely as part of the underlying disease process. Pet. Ex. 58 at 6.

[69] Tramadol is "an opioid analgesic used for the treatment of moderate to moderately severe pain following surgical procedures and oral surgery; administered orally." *Dorland's* 1920.

[70] Records of the complete hospital stay are contained in Pet. Ex. 82. Records of this hospitalization are also included in Pet. Ex. 88.

On July 19, 2018, petitioner was hospitalized for a "CIDP flare." Pet. Ex. 59 at 1. He had not received IVIG for six months. *Id*. at 19. He reported that testing at Weill Cornell showed generalized sensorimotor polyneuropathy with demyelinating features, but he was advised he needed to be treated in his home state of California. He received five days of IVIG and was discharged feeling much better. Pet. Ex. 59 at 2.

Petitioner presented to Dr. Belsky for a Medicare Annual Wellness visit on August 6, 2018. Pet. Ex. 106 at 614. Dr. Belsky documented idiopathic peripheral neuropathy with generalized weakness, no longer on corticosteroids, followed by neurology; uncontrolled Type 2 diabetes mellitus with long-term insulin use followed by an endocrinology; anxiety and depression treated with Zoloft;[71] anemia with thalassemia trait and malabsorption due to GI bleed. Weight loss was discussed. *Id*. at 614-15.[72]

Petitioner returned to Dr. Knox on August 10, 2018. His hip replacement and visit to Cornell were noted and that he was told he has CIDP. He reported receiving five days of IVIG in July 2018. On that day he complained of "firing" in his arms and hands, vibrating feelings, numbness in his fingers, and urgent need to urinate. Pet. Ex. 99 at 1. Neurological examination was normal. "Ankle and knee reflexes are trace but present. Brachioradialis biceps and triceps are 1+. Maybe a slight 5 weakness of right toe curling, maybe slight difficulties pinprick sensation in the top of the right toe, vibration 7 seconds". *Id*. at 5. Weak fingers with hand numbness. It "must be something else" if not CIDP. Most commonly, it would be something associated with the neck, but an EMG showed only carpal tunnel syndrome. Petitioner was referred to orthopedics for carpal tunnel surgery. Dr. Knox again documented that the lower back stimulator needed to be removed so an MRI could be done of his lumbar spine. Petitioner demonstrated "give way weakness,"[73] and the need for petitioner's records from Cornell. *Id*. at 10.

On September 21, 2018, petitioner presented to another neurologist, Dr. Xiong at UC Davis. Pet. Ex. 65 at 13. He reported a history of weakness and numbness in all extremities since a 2013 flu shot. He received the shot and felt like he was getting a cold, then his arm and hand were painful. The next day his hip joints hurt and a few days later he felt weak in the feet. About 3 weeks later he could not get up from the toilet and had difficulty raising his arm overhead; he felt pins and needles in his hands and his toes and legs were tingling. He went to the hospital, was diagnosed with GBS, treated with IVIG, and was back to normal. Then, 47 days later, he had similar symptoms and was treated with IV steroids. About 30 days after that, his feet and hands felt numb, and he had difficulty breathing. He received IVIG for five days and improved. He was diagnosed with CIDP and received high dose steroids for a year. He received IVIG for one to two years until it was stopped over 8 months ago. He felt worse without it. He had a right hip replacement in May 2018 and was hospitalized in July 2018 for weakness and numbness, received five days of IVIG, and felt 60 percent better, but started to decline a month later. He took Methotrexate and Imuran for a time in 2015 and 2016 without benefit. He saw Dr. Latov in March

[71] Zoloft is a brand name for preparations of sertraline hydrochloride. *Dorland's* 2061. Sertraline hydrochloride is a "selective serotonin reuptake inhibitor, used to treat depressive, obsessive-compulsive, and panic disorders; administered orally. *Dorland's* 1671.

[72] This record makes no reference to petitioner using any supportive aids to walk.

[73] While give way or giveaway weakness is often attributed to a patient who is not fully cooperating, is hysterical, malingering, or has conversion disorder, Dr. Chaudhry indicated that it can also be a result of pain or issues with balance. Tr. 210-11, 386.

2018, who recommended IVIG again, but Dr. Knox and Dr. Katz disagreed. *Id*. Dr. Xiong's impression was sensorimotor neuropathy with unclear underlying etiology. He noted that diabetes could contribute partially to his neuropathy, but it would be unusual for it to fluctuate. After reviewing the records, Dr. Xiong's working diagnosis was CIDP, but he stated it would be challenging to resume IVIG given his renal insufficiency while on IVIG. *Id*. at 20.

Dr. Latov wrote an addendum to his March record on September 26, 2018, "EMG and NCS on 6/21/18 showed changes consistent with a demyelinating polyneuropathy. Impression/Plan: He has a demyelinating polyneuropathy, probable chronic inflammatory demyelinating polyneuropathy (CIDP), although other causes of demyelinating polyneuropathy including MAG neuropathy, POEMS syndrome, or CMT1 should also be considered." Pet. Ex. 63 at 4.

Another EMG/NCS study ordered by Dr. Xiong and done at UC Davis on November 14, 2018 showed abnormal findings consistent with moderate motor and sensory peripheral neuropathy with both demyelinating and axonal features and superimposed carpal tunnel syndrome. Pet. Ex. 65 at 25.

Petitioner returned to Dr. Xiong on November 16, 2018 and was noted to be weaker. He wanted to know if he could start IVIG or Rituximab. Pet. Ex. 65 at 26. Dr. Xiong ordered IVIG with follow up in three months. Pet. Ex. 65 at 33-34.

g. 2019

Additional records were filed after the hearing, including records from physical therapy to improve mobility and make petitioner more independent with activities of daily living. Pet. Ex. 81 at 6. The physical therapy assessment included signs and symptoms suggestive of general weakness in all extremities but worse proximally, with significant static and dynamic balance deficits and significant sensation loss in all extremities. *Id*. at 27.

At a visit on February 8, 2019, Dr. Xiong noted "poor effort in exam" but normal muscle tone and bulk at his neck and appendages without fasciculations. He had mild action tremors in the right hand, but "FTN (Finger to Nose) intact bilaterally." He displayed difficulty rising and swing upon standing. Pet. Ex. 107 at 5. Dr. Xiong commented that the testing done at Cornell, Dr. Latov was significantly different from all the testing performed since 2013. There was sensory level loss below the neck, distal more than proximal weakness, and elements of poor effort. He had decreased/absent ankle reflexes with "otherwise preserved DTR (Deep Tendon Reflexes)." *Id*. at 9-10. "Diabetes could contribute to his peripheral neuropathy and possible inflammatory/immune-mediated etiology cannot (sic) completely rule out at this time. It is unusual for diabetic neuropathy to have significant fluctuation of symptoms and signs." *Id*. His slightly elevated IgA level in September 2018 could be related to IVIG treatment in July 2018. "Repeated EMG showed both demyelinating and axonal loss features, but it did not meet the EFNS electrophysiological criteria of CIDP." Hospital records show elevated CSF protein in 2013 and 2014, "but he was dx with diabetes before 2013. Therefore, CSF protein elevation could be partially related to diabetes and IVIG infusions at that time." He reported wetting himself, which raised concern of possible myelopathy. Petitioner refused MRIs of the brain and cervical spine to

rule out a possible CNS lesion. He refused repeat lumbar puncture and CSF studies and requested a second opinion with a neuromuscular specialist. *Id*.

On November 14, 2019, petitioner presented to Dr. Lin, a neurologist, for "unclear neuropathy diagnosis and DM that presents for second opinion on neuropathy." Pet. Ex. 107 at 11. Dr. Lin appears to have taken the history from Dr. Xiong's records, as it is identical. Petitioner thought he was seeing a neuromuscular specialist, not another neurologist. Petitioner reported his weakness had worsened, he was not on any immunomodulating treatment and was not receiving IVIG, PLEX, or Rituximab, but thought it would be therapeutic. He acknowledged the diagnostic uncertainty but felt IVIG was beneficial. He claimed it was discontinued due to complications with his kidney function, which was now back to normal, though his file did not confirm that. Petitioner reported a willingness to receive IVIG again, even it meant potential complications. Pet. Ex. 7 at 13. Dr. Lin's examination showed minor decrease in muscle strength, intact reflexes except at the Achilles, no ankle clonus, decreased sensation to pinprick from around C5 and below bilaterally, decreased vibration sensation at the thumbs, and absent sensation at B/L hallux. Finger to nose was without dysmetria or tremor B/L; heel to shin was without dysmetria B/L; rapid alternating movement was without dysdiadochokinesia B/L; and Romberg test was negative. Pet. Ex. 107 at 15. Dr. Lin's impression was neuropathy of unclear etiology, possibly with some contribution from diabetic neuropathy. Petitioner was referred to a neuromuscular disorders clinic and directed to discuss the possibility of restarting IVIG with them. *Id*. at 18.

Petitioner received an email thereafter that UC Davis did not have a specialist in CIDP, but Dr. Belsky could refer him to Stanford or UCSF. Pet. Ex. 107 at 20.

h. 2020

Updated medical records from 2020 and 2021 were filed on November 17, 2021. These records were not certified, and Petitioner's Exhibit 112 was printed from the website "myhealthonline.sutterhealth.org".

Petitioner was seen by Dr. Carroll on November 18, 2020 for chronic iron deficiency/anemia. His diagnoses included CIDP, DM, hypertension, and iron deficiency. Pet. Ex. 112 at 1. Dr. Carroll planned to continue with intravenous iron supplementation quarterly and keep ferritin greater than 100. Petitioner was encouraged to see his neurologist regarding his severe pain. His diabetes was noted as "suboptimally controlled." *Id*. at 2.

i. 2021

Petitioner participated in a video visit with Dr. Lai on March 5, 2021 for an acute flare of CIDP. Dr. Lai noted petitioner responded to IVIG previously and may need to present to the ER if things worsen. Pet. Ex. 112 at 4.

On April 17, 2021, petitioner presented to the ER with two days of right lower leg redness and reported infections in the same leg in the past. Pet. Ex. 112 at 6.[74]

[74] This record appears incomplete.

Petitioner presented to Dr. Belsky on April 22, 2021 for a routine examination. He complained of fatigue, weakness, and joint inflammation and was referred to rheumatology. Pet. Ex. 112 at 11-12. On examination, he was sitting in a wheelchair in no acute distress. He was noted as "[a]ble to stand with effort. Slow movements. Give away weakness in all muscles to upper and lower extremities. Hands and feet all warm and symmetrically edematous." There was no sign of cognitive impairment. *Id*. at 14. Lab results for that date were negative for ANA and CCP antibody IGG but showed elevated CRP and ESR. *Id*. at 27, 29, 31, 33. X-rays of his hands and feet showed inflammation. *Id*. at 36-39. Dr. Belsky discussed the acute inflammatory changes in petitioner's hands and feet with Dr. Knox. *Id*. at 11.

On May 25, 2021, petitioner presented to rheumatologist Dr. Zohuri, who documented that petitioner refused to put on a gown for the examination, expressed frustration that this was his third rheumatologic examination, and wanted IVIG treatment. Pet. Ex. 112 at 43. Dr. Zohuri took a full history noting there was no clear diagnosis. *Id*. The review of symptoms for rheumatologic process was negative and petitioner's biggest complaint was weakness and sensory loss. *Id*. at 42. Dr. Zohuri noted that ANA and Lyme testing were negative, polymyositis was unlikely because he had global weakness rather than proximal muscle weakness, and he had elevated IgA at one point, probably due to IVIG treatment at the time. There was no rheumatologic etiology found to explain his elevated ESR/CRP, but he had elevated inflammatory markers during his "GBS diagnosis/possible CIDP/demyelinating process 'flare'", which was the most plausible reason for the current elevated markers. His right hip replacement pathology showed all degenerative changes and nothing inflammatory, supporting a lack of inflammatory arthritis. Dr. Zohuri felt the issue was largely neurologic and his plan was to "check ANCA [antineutrophil cytoclasmic antibodies][75] and double check with neuro if a muscle biopsy will be high yield." Pet. Ex. 112 at 43.

Petitioner was admitted to the hospital on June 21, 2021 with generalized weakness, swelling in the bilateral extremities, joints of the shoulder, and hand, and severe pain. There was concern for cellulitis and CIDP flare. Empiric antibiotics improved the swelling of his legs, but his weakness persisted. Neurological consult was ordered. The neurology assessment noted "[i]t is unclear whether pt has CIDP, and ultimately he will need to see a neuromuscular specialist again for another opinion since there have been differing opinions among the neuromuscular [physicians] that he has seen." His presenting complaints were not consistent with exacerbation of CIDP. Pet. Ex. 112 at 142-43. Neurology felt it was inflammatory arthropathy rather than a CIDP flare and did not think IVIG would help though petitioner requested it. Prednisone was started and his swelling and pain improved within a few days, but petitioner advised that he benefits more from IVIG than prednisone long term. *Id*. at 166. He was encouraged to follow up with a neuromuscular specialist for this issue. *Id*. at 167. His diabetes was poorly controlled during his hospitalization. *Id*. at 154. He was discharged on June 24, 2021. *Id*. at 170.[76]

On October 4, 2021, petitioner presented to Dr. Shaoulian at the Neurology Muscular Dystrophy and Neuropathy Institute with an eight-year history of CIDP initially diagnosed as GBS

[75] Antineutrophil cytoclasmic antibody ("ANCA") is a blood test used to assist in the diagnosis of Wegener granulomatosis, a regional systemic vasculitis in which the small arteries of the kidneys, lungs, and upper respiratory tract are damaged by a granulomatous inflammation. *Mosby's* 79.

[76] The record routinely documents petitioner's refusal to have his leg braces brought to the hospital, adhere to a diabetic diet, or to engage in physical therapy unless he received IVIG. Pet Ex. 112 at 105, 111, 112.

and treated with steroids beginning 18 days after a flu vaccine. He was diagnosed with CIDP after a recurrence 47 days later. He presented with poor balance, a history of falls, use of a cane, walker and a wheelchair for short and long distances, respectively. Pet. Ex. 111 at 6. The assessment after examination was weakness in all extremities with loss of vibration sense in the distal lower and upper extremities and decreased pain sensation in the distal lower extremities. The 2018 EMG/NCS from Cornell was read as generalized sensorimotor polyneuropathy with demyelinating features. Labs from Sutter in 2017 and 2018 were negative for monoclonal bands and he had multiple levels of foraminal narrowing. The diagnosis on that date was hyperesthesia; difficulty walking, not elsewhere classified; and weakness. *Id*. at 7.

On November 8, 2021, petitioner returned to Dr. Shaoulian for EMG/NCS testing using a bipolar needle. "Different muscles were tested to reflect different root and nerve distributions. The EMG was normal." Pet. Ex. 111 at 1. The conclusion documented "significant delayed F-waves and decreased CMAP velocities. The study is consistent with demyelinating sensory motor neuropathy." *Id*. at 1. Dr. Shaoulian also conducted an examination on that date, noting weakness in all extremities, with loss of vibration sense in the distal lower and upper extremities and decreased pain sensation in the distal lower extremities. *Id*. at 5. Dr. Shaoulian wrote that the EMG/NCS "is consistent with a demyelinating sensory motor neuropathy. Based on the patient's history, exam and workup he has CIDP." He ordered IVIG for two months. *Id*.

C. Testimony of Petitioner Harvard Davis[77]

Petitioner appeared in a wheelchair at hearing.[78] Petitioner earned an undergraduate degree from Queens College in business management and associate degree from El Paso University in human resource management. Tr. 5. He was in the marine business for over thirty years as a mechanic, rigger, installation and set-up mechanic, and a master tech authorized to train people and bring them into the business. Tr. 6. An average day in the industry includes putting the right product in the customers' hands based on their needs, unloading shipments, stacking, counting, inventorying products, and interacting with customers. Tr. 6-7.

According to petitioner, he worked at a West Marine store until 2007 when he took a job with Cope McPhetres. From 2010 until the end of 2011, he worked for Goodwill. He was then unemployed until he returned to West Marine in March of 2012, where he was working at the time of his vaccination in August of 2013. Tr. 86-87. Later, petitioner stated that Cope McPhetres went bankrupt in 2008 or 2009 and he was then on unemployment until he was hired by Goodwill Industries at the beginning of 2010. He was dismissed from employment by Goodwill in 2011 and rehired by West Marine in 2012. Tr. 123-24. When asked, petitioner agreed that he was dismissed

[77] Petitioner did not file an affidavit in support of his petition. The statute and the rules require the documentation supporting the claim be filed with the petition. 42 U.S.C.A. § 300aa-11(c); RCFC, Appendix B, Vaccine Rule 2. The Vaccine Rules specifically state that "if the required medical records are not submitted, the petitioner must include an affidavit detailing the efforts made to obtain such records and the reasons for their unavailability. If petitioner's claim does not rely on medical records alone but is also based in any part on the observations or testimony of any person, the petitioner should include the substance of each person's proposed testimony in a detailed affidavit(s) supporting all elements of the allegations made in the petition." RCFC, Appendix B, Vaccine Rule 2(c)(2)(B)(i)-(ii).

[78] During his testimony, Dr. Chaudhry referenced petitioner using a wheelchair at hearing. Tr. 352.

from Goodwill in 2010 and was out of work for around 15 months until he was rehired by Marine West in March of 2012. Tr. 125.[79]

Petitioner described himself as lively, "on the move", working, and able to handle his social life. His health was "perfect" prior to the August 19, 2013 flu vaccine. Tr. 8. He went wakeboarding, played basketball, snowboarded, did offshore boating, and "just had lots of fun." Tr. 10. Petitioner agreed he had a "mini stroke" in 2010, high blood pressure, spinal surgeries after a motor vehicle accident, and always had some pain, but could put it out of his mind. Tr. 9.

Petitioner acknowledged having diabetes and not monitoring his blood sugar levels but controlled it by watching his weight and taking Glipizide.[80] Petitioner acknowledged that Dr. Van sent him to a class to learn to monitor his diabetes, but he did not go due to loss of insurance and never got the monitoring equipment. Tr. 88-92. Petitioner testified that he did not see a doctor in 2012 because he did not have insurance. Tr. 15-16. Petitioner stated CVS filled all his prescriptions, but he did not see any physicians during the period he was uninsured. Tr. 15-19; 92. He stated he received a flu vaccine every year and paid for the 2012 flu vaccine himself. Tr. 20.

Petitioner testified that the severity of his diabetes was due to the steroids needed following his flu vaccine and it was Dr. Belsky who sent him to the class to learn how to control his diabetes. "My levels were not that outrageous, Ma'am. And I didn't have any – I didn't have any thirst. I didn't have any – I had weight gain. I had gained a lot of weight, and that was one of the things Dr. Van really counseled me about was, Mr. Davis, you need to lose weight." Petitioner ultimately agreed that it was Dr. Van in 2011 who told him he needed to go to class to learn to monitor his diabetes and ordered all the equipment, but he did not go. Tr. 128-29. Petitioner stated his endocrinologist Dr. McMullen told him in 2017 that the Solu-Medrol treatment drove his numbers so high he could no longer control his diabetes with pill therapy. Tr. 129-131.

Petitioner stated he provided his medical history to Dr. Hopkins at his first visit on April 24, 2013. He acknowledged that the records note multiple medical problems including poorly controlled diabetes with neuropathy and "bilateral foot pain from which he limps around." He disagreed that he had bilateral foot pain or that he limped around but rather had a heel callous that caused pain and when he got rid of the callous, he felt great, was back to working, climbing ladders, going to car shows, playing ball, and exercising on the treadmill. Tr. 10-11, 13-14. Dr. Hopkins ran a pin over his feet and said he had neuropathy, but he did not know what neuropathy was prior to this visit. He denied his "feet hurt that much." Tr. 15, 96, 130-31; Pet. Ex. 9 at 6. According to petitioner, Dr. Hopkins prescribed Gabapentin but he never filled the prescription or took it. Tr. 11. Petitioner agreed that Dr. Hopkins's record also documented decreased sensation in his feet, chronic lower back and hip pain, and arthritis in his back. Tr. 97-98; Pet. Ex. 9 at 7-10.

[79] Petitioner could not recall when he stopped working after his August 2013 vaccination, but believed it was in 2015. Tr. 102.

[80] A June 2011 medical record shows petitioner's diabetes to be uncontrolled with high blood sugar levels. He was scheduled by his physician to attend a maintenance basics class and was to email blood sugar readings to his then-PCP, Dr. Van. Petitioner acknowledged the content of the record but stated he did not go to the class because Goodwill cut off his insurance. Pet. Ex. 4 at 116, 119.

Petitioner described the events following his August 19, 2013 flu vaccine. It was Monday, his day off, and he was doing errands. He stopped at work, was told the vaccine came in and that the company pays for it, so he got the vaccine. He finished his errands and went home. He was doing work at home, felt "blah", started getting stiff, and his shoulder where he got the shot started to hurt "because the shot was on the top of his arm". Tr. 20-21. He finished his work and laid on the couch. He then contacted another employee to switch days off so he could stay home on Tuesday. He laid on the couch all day Tuesday and went to bed early. He felt good on Wednesday, went to work, and was able to function "pretty good." Tr. 21-23.

Petitioner was asked about the history he provided at the ER, which included right leg pain the day after the vaccination, being unable to work for three days, and worsening symptoms in the weeks that followed. Petitioner stated that he had symptoms for weeks before he presented to the emergency room. Tr. 23-27; Pet. Ex. 3 at 8. Petitioner then stated he did not feel well immediately after the vaccination, but it was "different kind of symptoms" than those he experienced the day of the car show, September 14. Tr. 27. On that day, when he awoke at 5:00 am, he had difficulty getting out of bed because the side of his right leg was numb, and he had hip pain. He could not walk straight or get his leg to do what he wanted it to. Tr. 27-28. His "hands were like flippers", he could not grip anything and had difficulty opening his car door and getting into the car. He drove to the car show palming the steering wheel. Tr. 28-29. When he arrived, someone had to help him open the car door and get out of the car. Tr. 30. He stated he was confronted by security, who thought he was intoxicated because he could not walk straight. Security drove him back to his car and he left the show around 1:30 pm. Tr. 31-32. According to petitioner, he could not control his body, so he drove home slowly, unable to apply pressure to the gas pedal. Tr. 33.

Petitioner stated he went to work the next day but was unable to get the keys in the door, kept dropping things, tried to run but couldn't, couldn't get up the ladder to get product, couldn't pick up coins at the cash register, and could not separate paper money. Tr. 33-36.

Petitioner testified he reported all of this to Dr. Hopkins on September 17, 2013 and has no idea why these complaints were not in the record. Tr. 36-37. He recalled that Dr. Hopkins knew he had arthritis in his back and asked what was different about the past few weeks. Petitioner told him he had gotten his flu vaccine and Dr. Hopkins told him he had Guillain-Barre, a reaction to the flu shot, and gave him a steroid pack. Tr. 36-38.

Petitioner stated the next fifteen days were "horrible" and he had a hard time functioning. The steroid pack worked for a day or two but the "symptoms came back like a vengeance." Tr. 37. After he finished the steroids, he could not do anything; he could not unload boxes, raise his hands over his head, or operate the cash register. Tr. 39-40.

Petitioner stated when he awoke on September 29, 2013, he stumbled to the bathroom, was unable to get up from the toilet, and had to crawl into his bedroom and call his roommate for help getting to his car. He drove to the hospital and asked someone in the parking lot to get help, and a man took him into the emergency room in a wheelchair. Tr. 40-42.

Petitioner stated he told Dr. Mahmood that the day after the flu vaccine, he could not lift himself up and his legs felt weak, but he did not tell him he could not walk. He felt "icky" but not

like the "crippling event like it was on the 14th". Tr. 42-44. Petitioner stated he was referring to September 14, 2013, when he told Dr. Mahmood about the shoulder pain, tenderness, weakness, and inability to open the car door or reach overhead. Tr. 45.

When asked again later if he reported generalized aches in his lower extremities the day after the flu vaccine that worsened in the following days to Dr. Mahmood, he responded that he was trying to explain that he got a flu shot a few weeks prior and it was a few days after the shot that he started feeling differently. Tr. 118-19; Pet. Ex. 3 at 26-28. He believed the hospital already had his history. Tr. 47. When he reported joint pain, he meant his knuckles hurt when he tried to close his hands, his shoulders hurt when he raised his hands overhead, his hips hurt, his legs were heavy, and he could not lift them or straighten his knees. These symptoms were, "Overwhelming. Debilitating. Nonfunctioning. Allowing me not to function or do anything. No strength." He had none of these symptoms before the flu vaccine. Tr. 50-51. He had no spinal pain before the vaccination and did not take pain medication after 2010 or 2011. Tr. 51. Petitioner would only admit that he took Vicodin and Tramadol for back pain prior to the flu vaccine when confronted with a record from 2012. Tr. 92-95; Pet. Ex. 8 at 38-39.

Petitioner stated Dr. Mahmood told him he had GBS from the flu vaccine and had to report it to the CDC. Dr. Mahmood did not tell him that GBS would not explain the muscle tenderness or soreness. Tr. 42, 47, 99.

According to petitioner, he went home and back to work after five days of IVIG and some physical therapy. Tr. 48, 51. IVIG was the "miracle drug"; he had his strength back, he could run and do whatever he wanted. Tr. 52. About a month later, when he tried to help a customer at work, he could not reach his arms overhead or use the ladder because his right leg was not working correctly and his "foot just kept dropping." Tr. 52-54. Over the next week, it accelerated and on October 29, 2013, he got out of bed, "fell right down on my face," and "had no legs." Tr. 54. He called his neighbor to take him to the hospital. Tr. 55. He told the doctor in the ER about his symptoms, referencing the incident at the store two days prior when he could not climb a ladder. Tr. 56-57; Pet. Ex. 3 at 69.

According to petitioner, he saw Dr. Mahmood again who ran several tests and put him on Solu-Medrol and steroids. The spinal tap came back higher than it should be, but everything else was normal. Tr. 58-60. Dr. Mahmood said his symptoms were unclear and he may have myopathy that was steroid responsive. Petitioner "got lost in the science of it". Tr. 99-100. After the Solu-Medrol, he felt great again for 30-40 days, but then he went to Dr. Hopkins's office because he couldn't breathe, and they called an ambulance. Tr. 60.

Petitioner stated Dr. Mahmood wanted an MRI of his back, which could not be done because he had a stimulator implanted in his back since 1995. He did not recall any doctor other than Dr. Knox saying his symptoms were related to diabetes, but he agreed he "still wasn't monitoring" his diabetes at that time. Tr. 61.

Petitioner stated Dr. Seminer, his neurologist, thought he had CIDP and gave him steroids. Tr 62. He recalled an EMG that showed a little abnormality but was overall normal. IVIG was

started. Tr. 62-63; Pet. Ex. 3 at 113.[81] When presented with the EMG results and Dr. Seminar's record, petitioner agreed Dr. Seminar did not think the study supported GBS or CIDP. Tr. 101; Pet. Ex. 3 at 106-07.

Petitioner stated he saw Dr. Knox for the first time some time in 2014 when he was taken by ambulance to the hospital after having trouble breathing. Tr. 65. Dr. Knox said he had CIDP and did an EMG. He started him on steroids and Toradol, then Benadryl, then IVIG several weeks later, which gave him bad headaches and he could not function at work. Tr. 66-67, 101.

Petitioner stated Dr. Knox got frustrated that the nerve conduction studies did not show anything but told him he had CIDP, an autoimmune disease that attacks the nerves. He was giving him IVIG to help the nerves, but he stopped it due to the headaches. Tr. 67. Petitioner agreed that Dr. Knox's records in June and July 2015 state, "possible primary muscle disorder" and that Dr. Knox wanted a muscle biopsy. Tr. 103-04; Pet. Ex. 20 at 46-47. He further agreed that at his next visit on October 1, 2015, Dr. Knox wrote "possible myopathy but with numbness places this into the CIDP or similar; could be myelopathy, but has no spasticity?" Tr. 104; Pet. Ex. 18 at 7. Petitioner agreed that Dr. Knox was questioning the CIDP diagnosis in 2015, "He was looking for all types of other findings, and blood tests showed that, too." Tr. 105.

Petitioner recounted that Dr. Knox took him to a dinner in 2016 with other doctors and nurses to discuss his case and why his EMGs were normal. There was a slideshow and handouts, and the discussion used his name. Tr. 68, 71-73; Pet. Ex. 91. The doctors spoke about how the small nerves could be the problem. Dr. Muley[82] told him that the nervous system is like wires; myelin is insulation that covers the wire if there is a break somewhere and IVIG coats and rebuilds that. Tr. 69-70. Dr. Muley told him Rituxan may be an option, as it has worked on patients with CIDP. Tr. 70. He filed the only paperwork he received from the dinner as Pet. Ex. 91. Tr. 71-73. He never heard anything further after the dinner and did not receive any write up about his case. Tr. 121-22.

Petitioner stated insurance stopped his IVIG treatment in 2016 but it resumed after an appeal via email, but he could not find the emails. Tr. 74. Dr. Knox stopped his treatment again in 2017 after an incident at the infusion center requiring hospitalization. Tr. 75.

Petitioner stated Dr. Knox sent him to Dr. Katz in San Francisco for a second opinion. Tr. 77. Dr. Katz did not perform an examination, spent the visit searching for his file and discussing the photographs on his wall of New York, and said "You look too healthy to have CIDP". Tr. 77-79. Petitioner could not explain how Dr. Katz's record reflected an examination and findings other than to say "I don't know. I just remember sitting there talking with him, you know. I've had other exams that I – that were so much more thorough than what he gave me, and just sitting and talking to me and then not finding my records and talking about a picture on the wall…" Tr. 105.

[81] Petitioner was asked if he had any independent recollection of the events he was testifying to and admitted that he did not but was reading from the records. He was asked to put away his notes.

[82] Petitioner recalled this doctor's name as "Mule", but the record filed from the event, Pet. Ex. 91, lists Dr. Muley as the speaker.

Petitioner testified to only seeing Dr. Knox once more after his visit with Dr. Katz because Dr. Knox stopped his treatment, stating his problems were due to his diabetes. Petitioner told Dr. Knox that he never needed insulin for his diabetes until he was treated with steroids, but he now needed insulin. Dr. Knox wanted him to have surgery to remove the stimulator and for carpal tunnel and scheduled it twice. Tr. 79-81. At that point, he lost confidence in Dr. Knox and sought another opinion from Dr. Latov in New York City. Tr. 79-81, 105-06. Later, petitioner conceded that he continued to see Dr. Knox in 2017 and until after he saw Dr. Latov in 2018. Tr. 107. Updated records filed show that Dr. Knox was still listed as petitioner's physician in 2021. Pet. Ex. 112 at 11.

Petitioner described Dr. Latov's examination as an hour and half to two hours long. Dr. Latov told him he should have been on Rituxan rather than IVIG and ordered EMG testing, which showed "sensorimotor polyneuropathy, and there was demyelination." Tr. 82. Dr. Latov told him he had CIDP or "Poems or something" and a treatment plan needed to be started in California. Tr. 83. Petitioner agreed he had not received IVIG for about eight months at that time. Tr. 84. Petitioner later agreed that Dr. Latov wrote generalized weakness and sensory loss of unclear cause after his visit and that he only saw Dr. Latov once. Tr. 107-09; Pet. Ex. 58 at 4. The addendum written in September of 2018 was not associated with a visit. Tr. 108-09; Pet. Ex. 63. He then stated he saw Dr. Latov twice, in March and in June, when the EMG was performed, but did not speak to him after the EMG and never saw him again. Petitioner stated Dr. Latov did not communicate with Dr. Knox or any other doctor in California about Rituxan; Rituxan was only discussed between Dr. Latov and petitioner at the March visit. Tr. 109-112. Petitioner was covered by Medicaid/Medi-Cal at that time. Tr 112-14.

Petitioner stated he saw Dr. Brown at the hospital in July 2018 and then Dr. Xiong at UC Davis in September and November 2018. Tr. 114. He received physical therapy from October to December 2018. Tr. 115; Pet. Ex. 63. Petitioner stated he had another EMG in the fall of 2018 and NeuTrexin[83] was suggested. Tr. 115-16; Pet. Ex. 64 at 13; Pet. Ex. 81 at 1. At the time of the hearing, petitioner was not receiving any treatment for his neurological issues and had not had an IVIG infusion or IV steroids since July of 2018. Tr. 116-17.

Petitioner denied that he was self-prescribing prednisone or that he was warned to stop taking it by his doctors as reflected in his records. He claimed he only had a half a bottle from his visit with Dr. Seminer and only took it between treatments. He had no idea where the information in the medical record came from. Tr. 125-26.

Petitioner disagreed that his diagnosis of CIDP was uncertain. Tr. 119-120. "What I do believe is that because my tests are irregular, the EMG, that's what they're basing so much of this on", but "they've continued to say CIDP". Tr. 120. He agreed that Dr. Latov's inclusion of other differential diagnoses, including CIDP, would be the "right thing to possibly do if . . . you've only seen me one time or two times." Tr. 120.

Petitioner recalled that Dr. Scalapino told him it was unclear whether he had CIDP, CIDP related to diabetes, or some other peripheral neuropathy. At that time, he had "full blown" diabetes

[83] NeuTrexin is a brand name for trimetrexate, which is "a folic acid antagonist structurally related to methotrexate; it competitively inhibits dihydrofolate reductase . . . administered intravenously as the glucuronate salt." *Dorland's*

with blood sugar levels of 600 due to steroids. No one ever discussed the progression of diabetic neuropathy with him. Tr. 126-27; Pet. Ex. 89 at 94.

D. Petitioner's Declaration

Following the hearing, petitioner filed a declaration addressing the muscle biopsies that were ordered. Pet. Ex. 110. Petitioner declared that Dr. Knox's "records were repetitive. In other words, he did not recommend a biopsy on several occasions to me. Rather, he discussed it with me a couple of times, originally, and again, after I explained that I had not obtained one due to lack of insurance. The record in March 2016, indicated 'but can wait for now.'" *Id*. at 2. Petitioner further declared that neither Dr. Knox nor Dr. Latov ordered or recommended a muscle biopsy after he had insurance in April 2016. *Id*. Petitioner listed references in the record where muscle biopsy was documented but denied that the muscle biopsies were mentioned to him when he saw Dr. Knox and Dr. Xiong in 2017 and 2018. *Id*. at 1-2. Petitioner declared he would have done it if he thought it was vital to his health. *Id*. at 2.

III. The Experts

A. Petitioner's Expert, Dr. Lawrence Steinman[84]

Dr. Steinman is a neurologist at Stanford University. Pet. Ex. 75. He graduated from Dartmouth College with a degree in physics and attended Harvard Medical School, where he also completed a fellowship in chemical neurobiology. During his residency at Stanford University Hospital, he completed a fellowship in chemical immunology. He has been a professor at Stanford since 1980, with appointments in neurology, neurological sciences, pediatrics, and genetics. *Id*. Dr. Steinman is board certified in neurology and has published hundreds of articles on the immunological aspects of neurologic disease. He is considered an expert on multiple sclerosis (MS) and holds several patents for therapies used to treat MS. Dr. Steinman's most updated CV was filed on February 5, 2019. Pet. Ex. 75.[85]

[84] Dr. Steinman is a brilliant doctor, who is well known to the court and has for many years testified on behalf of petitioners in the Vaccine Program. He has however in recent years been cautioned about his conduct with the court, counsel, and witnesses. He is once again cautioned here for digressing from his role as an expert to criticize others particularly those who questioned the diagnosis of CIDP. Tr. 232, 238, 241. Dr. Steinman made his customary comments that he was not an advocate for either side but simply presenting the science. Tr. 176, 242-43, 252. However, many of his digressions were argumentative and adversarial. *See D.G. v. Sec'y of Health & Hum. Servs.*, No. 11-577V, 2019 WL 2511769, at *189 (Fed. Cl. May 24, 2019) (criticizing Dr. Steinman's behavior toward respondent's expert and noting that his conduct made him an advocate), *Caredio v. Sec'y of Health & Hum. Servs.*, No. 17-0079V, 2021 WL 4100294, at *15 n.13 (Fed. Cl. July 30, 2021), review denied, No. 17-79V, 2021 WL 6058835 (Fed. Cl. Dec. 3, 2021) (noting that Dr. Steinman's "various asides" greatly detracted from his credibility), *Sanchez v. Sec'y of Health & Hum. Servs.*, No. 11-685V, 2020 WL 5641872 at *15 (Fed. Cl. Aug. 26, 2020), review denied sub nom. Sanchez by & through Sanchez v. Sec'y of Health & Hum. Servs., 152 Fed. Cl. 782 (2021) (citing several cases in which "[s]pecial masters have indicated that Dr. Steinman presents as an advocate for the petitioners who retain him."). Notably, his demeanor was appropriate and respectful during the continuation of the hearing in the fall of 2019.

B. Respondent's Expert, Dr. Vinay Chaudhry

Dr. Vinay Chaudhry received his Bachelor of Medicine and Bachelor of Surgery degrees from All India Institute of Medical Sciences. Resp. Ex. B. He is board certified in neurology, electrodiagnostic medicine, clinical neurophysiology, and neuromuscular medicine. Dr. Chaudhry has served as a professor of neurology at Johns Hopkins since 2004, where he has chaired the neurology credentials committee since 2009. He has acted as a consultant for respondent regarding cases in the Vaccine Program since 1999. Dr. Chaudhry is a member of the editorial board for the journal *Neurologist*. Dr. Chaudhry is an expert on electrodiagnostic studies as they relate to neuromuscular diseases. He has an active clinical practice and is involved in clinical research and teaching at the Johns Hopkins Hospital in Baltimore, MD. *Id*.

IV. Causation and Analysis

A. Legal Standard Regarding Causation

The Vaccine Act provides two avenues for petitioners to receive compensation. First, a petitioner may demonstrate a "Table" injury—i.e., an injury listed on the Vaccine Injury Table that occurred within the provided time period. 42 U.S.C. § 300aa-11(c)(1)(C)(i). "In such a case, causation is presumed." *Capizzano v. Sec'y of Health & Human Servs*., 440 F.3d 1317, 1320 (Fed. Cir. 2006); *see* § 13(a)(1)(B). Second, where the alleged injury is not listed on the Vaccine Injury Table, a petitioner may demonstrate an "off-Table" injury, which requires that the petitioner "prove by a preponderance of the evidence that the vaccine at issue caused the injury." *Capizzano*, 440 F.3d at 1320; *see* § 11(c)(1)(C)(ii). A petitioner need not show that the vaccination was the sole cause, or even the predominant cause, of the alleged injury; showing that the vaccination was a "substantial factor" and a "but for" cause of the injury is sufficient for recovery. *Pafford v. Sec'y of Health & Human Servs*., 451 F.3d 1352, 1355 (Fed. Cir. 2006); *Shyface v. Sec'y of Health & Human Servs*., 165 F.3d 1344, 1352 (Fed. Cir. 1999).[86] Petitioners are not required "to eliminate alternative causes as part of establishing [their] prima facie case." *Doe v. Sec'y of Health & Human Servs*., 601 F.3d 1349, 1357-58 (Fed. Cir. 2010); *see Walther v. Sec'y of Health & Human Servs*., 485 F.3d 1146, 1152 (Fed. Cir. 2007) (holding that a "petitioner does not bear the burden of eliminating alternative independent potential causes"). Once a petitioner has proven causation by preponderant evidence, "the burden then shifts to the respondent to show by a preponderance of the evidence that the injury is due to factors unrelated to the administration of the vaccine." *Deribeaux ex rel. Deribeaux v. Sec'y of Health & Human Servs*., 717 F.3d 1363, 1367 (Fed. Cir. 2013) (citing 42 U.S.C. § 300aa-13(a)(1)(B)).

To prove causation, petitioners must satisfy the three-pronged test established in *Althen v. Sec'y of Health & Human Servs*., 418 F.3d 1274 (Fed. Cir. 2005). *Althen* requires that petitioners show by preponderant evidence that a vaccination petitioner received caused his or her injury "by providing: (1) a medical theory causally connecting the vaccination and the injury; (2) a logical sequence of cause and effect showing that the vaccination was the reason for the injury; and (3) a showing of a proximate temporal relationship between vaccination and injury." *Id*. at 1278.

[86] The Vaccine Act also requires petitioners to show by preponderant evidence that the "residual effects or complications" of the alleged vaccine-related injury lasted for more than six months. § 11(c)(1)(D)(i). It is undisputed that this six-month requirement is satisfied in this case.

Together, these prongs must show "that the vaccine was 'not only a but-for cause of the injury but also a substantial factor in bringing about the injury.'" *Stone v. Sec'y of Health & Human Servs*., 676 F.3d 1373, 1379 (Fed. Cir. 2012) (quoting *Shyface*, 165 F.3d at 1352-53). Causation is determined on a case-by-case basis, with "no hard and fast per se scientific or medical rules." *Knudsen v. Sec'y of Health & Human Servs*., 35 F.3d 543, 548 (Fed. Cir. 1994). Petitioners are not required to identify "specific biological mechanisms" to establish causation, nor are they required to present "epidemiologic studies, rechallenge, the presence of pathological markers or genetic disposition, or general acceptance in the scientific or medical communities." *Capizzano*, 440 F.3d at 1325 (quoting *Althen*, 418 F.3d at 1280). "[C]lose calls regarding causation are resolved in favor of injured claimants." *Althen*, 418 F.3d at 1280.

Each *Althen* prong requires a different showing. Under the first prong, petitioner must provide a "reputable medical theory" demonstrating that the vaccine received can cause the type of injury alleged. *Pafford*, 451 F.3d at 1355-56 (citation omitted). To satisfy this prong, petitioner's "theory of causation must be supported by a 'reputable medical or scientific explanation.'" *Andreu*, 569 F.3d at 1379 (quoting *Althen*, 418 F.3d at 1278). This theory need only be "legally probable, not medically or scientifically certain." *Id*. at 1380 (emphasis omitted) (quoting *Knudsen*, 35 F.3d at 548). Nevertheless, "petitioners [must] proffer trustworthy testimony from experts who can find support for their theories in medical literature." *LaLonde*, 746 F.3d at 1341.

The second *Althen* prong requires proof of a "logical sequence of cause and effect." *Capizzano*, 440 F.3d at 1326 (quoting *Althen*, 418 F.3d at 1278). Even if the vaccination can cause the injury, petitioner must show "that it did so in [this] particular case." *Hodges v. Sec'y of Health & Human Servs*., 9 F.3d 958, 962 n.4 (Fed. Cir. 1993) (citation omitted). "A reputable medical or scientific explanation must support this logical sequence of cause and effect," *Id*. at 961 (citation omitted), and "treating physicians are likely to be in the best position to determine whether a logical sequence of cause and effect show[s] that the vaccination was the reason for the injury," *Paluck v. Sec'y of Health & Human Servs*., 786 F.3d 1373, 1385 (Fed. Cir. 2015) (quoting *Andreu*, 569 F.3d at 1375).

However, medical records and/or statements of a treating physician's view do not per se bind the special master to adopt the conclusions of such an individual, even if they must be considered and carefully evaluated. Section 12(b)(1) (providing that "[a]ny such diagnosis, conclusion, judgment, test result, report or summary shall not be binding on the special master or court."); *Snyder v. Sec'y of Health & Human Servs*., 88 Fed. Cl. 706, 746 n.67 (2009) ("there is nothing…that mandates that the testimony of a treating physician is sacrosanct—that it must be accepted in its entirety and cannot be rebutted"). As with expert testimony offered to establish a theory of causation, the opinions or diagnoses of treating physicians are only as trustworthy as the reasonableness of their suppositions or bases. The views of treating physicians should also be weighed against other, contrary evidence also present in the record – including conflicting opinions among such individuals. *Hibbard v. Sec'y of Health & Human Servs*., (100 Fed. Cl. 742, 749 (2011) (determining it is not arbitrary or capricious for a special master to weigh competing treating physicians' conclusions against each other), *aff'd*. 698 F.3d 1355 (Fed. Cir. 2012); *Caves v. Sec'y of Health & Human Servs*., 100 Fed. Cl. 119, 136 (2011), *aff'd*, 463 Fed. Appx. 932 (Fed. Cir. 2012); *Veryzer v. Sec'y of Health & Human Servs*., No. 06-522V, 2011 WL 1935813, at *17

(Fed. Cl. Spec. Mstr. Apr. 229, 2011), *mot. for review den'd*, 100 Fed. Cl. 344 (Sept. 29, 2011), *aff'd*, 475 Fed. Appx. 765 (Fed. Cir. 2012).

The third *Althen* prong requires that petitioner establish a "proximate temporal relationship" between the vaccination and the alleged injury. *Althen*, 418 F.3d at 1281. This "requires preponderant proof that the onset of symptoms occurred within a timeframe for which, given the medical understanding of the disorder's etiology, it is medically acceptable to infer causation-in-fact." *De Bazan v. Sec'y of Health & Human Servs*., 539 F.3d 1347, 1352 (Fed. Cir. 2008). Typically, "a petitioner's failure to satisfy the proximate temporal relationship prong is due to the fact that onset was too late after the administration of a vaccine for the vaccine to be the cause." *Id*. However, "cases in which onset is too soon" also fail this prong; "in either case, the temporal relationship is not such that it is medically acceptable to conclude that the vaccination and the injury are causally linked." *Id*.; *see also Locane v. Sec'y of Health & Human Servs*., 685 F.3d 1375, 1381 (Fed. Cir. 2012) ("[If] the illness was present before the vaccine was administered, logically, the vaccine could not have caused the illness.").

Finally, although this decision discusses much but not all the literature submitted by the parties, the undersigned reviewed and considered all the medical records and literature submitted in this matter. *See Moriarty ex rel. Moriarty v. Sec'y of Health & Human Servs*., 844 F.3d 1322, 1328 (Fed. Cir. 2016) ("We generally presume that a special master considered the relevant record evidence even though [s]he does not explicitly reference such evidence in h[er] decision."); *Simanski v. Sec'y of Health & Human Servs*., 115 Fed. Cl. 407, 436 (2014) ("[A] Special Master is 'not required to discuss every piece of evidence or testimony in her decision.'" (citation omitted)), *aff'd*, 601 F. App'x 982 (Fed. Cir. 2015).

B. The Expert Reports and Testimony

Establishing a sound and reliable medical theory often requires a petitioner to present expert testimony in support of his claim. *Lampe v. Sec'y of Health & Human Servs*., 219 F.3d 1357, 1361 (Fed. Cir. 2000). Vaccine Program expert testimony is usually evaluated by the factors for analyzing scientific reliability set forth in *Daubert v. Merrell Dow Pharm., Inc.*, 509 U.S. 579, 594-96 (1991).[87] *Cedillo v. Sec'y of Health & Human Servs*., 617 F.3d 1328, 1339 (Fed. Cir. 2010) (citing *Terran v. Sec'y of Health & Human Servs*., 195 F.3d. 1302, 1316 (Fed. Cir. 1999)).

The *Daubert* factors are usually employed by judges in the performance of their evidentiary gatekeeper roles to exclude evidence that is unreliable and/or could confuse the jury. In Vaccine Program cases, by contrast, these factors are used in the weighing of the reliability of scientific evidence proffered. *Davis v. Sec'y of Health & Human Servs*., 94 Fed. Cl. 53, 66-67 (2010) ("uniquely in this Circuit, the *Daubert* factors have been employed also as an acceptable evidentiary-gauging tool with respect to persuasiveness of expert testimony already admitted"). The flexible use of the *Daubert* factors to evaluate the persuasiveness of expert testimony has

[87] The *Daubert* factors for analyzing the reliability of testimony are: (1) whether a theory or technique can be (and has been) tested; (2) whether the theory or technique has been subjected to peer review and publication; (3) whether there is a known or potential rate of error and whether there are standards for controlling the error; and (4) whether the theory or technique enjoys general acceptance within a relevant scientific community." *Terran*, 195 F.3d at 1316 n.2 (citing *Daubert*, 509 U.S. at 592-95).

routinely been upheld. *See, e.g.*, *Snyder*, 88 Fed. Cl. at 742-45. In this case, as in numerous other Vaccine Program cases, *Daubert* has not been employed to determine what evidence should be admitted, but rather to determine whether expert testimony offered is reliable and/or persuasive.

Where both sides offer expert testimony, a special master's decision may be "based on the credibility of the experts and the relative persuasiveness of their competing theories." *Broekelschen v. Sec'y of Health & Human Servs*., 618 F.3d 1339, 1347 (Fed. Cir. 2010) (citing *Lampe*, 219 F.3d at 1362). However, nothing requires the acceptance of an expert's conclusion, "connected to existing data only by the *ipse dixit* of the expert," especially if "there is simply too great an analytical gap between the data and the opinion proffered." *Snyder*, 88 Fed. Cl. at 743 (quoting *Gen. Elec. Co. v. Joiner*, 522 U.S. 136, 146 (1997)); *see also Isaac v. Sec'y of Health & Human Servs*., No. 08-601V, 2012 WL 3609993, at *17 (Fed. Cl. Spec. Mstr. July 30, 2012), *mot. for review den'd*, 108 Fed. Cl. 743 (2013), *aff'd*, 540 F. App'x. 999 (Fed. Cir. 2013) (citing *Cedillo*, 617 F.3d at 1339). Weighing the relative persuasiveness of competing expert testimony, based on a particular expert's credibility, is part of the overall reliability analysis to which special masters must subject expert testimony in Vaccine Program cases. *Moberly v. Sec'y of Health & Human Servs*., 592 F.3d 1315, 1325-26 (Fed. Cir. 2010) ("[a]ssessments as to the reliability of expert testimony often turn on credibility determinations"); *see also Porter v. Sec'y of Health & Human Servs*., 663 F.3d 1242, 1250 (Fed. Cir. 2011) ("this court has unambiguously explained that special masters are expected to consider the credibility of expert witnesses in evaluating petitions for compensation under the Vaccine Act.").

Application of the *Althen* prongs reveals evidentiary support for petitioner's claim: (1) petitioner offered a persuasive or reliable medical theory; (2) the theory provided was applicable in part to the facts of petitioner's case; and (3) petitioner established a medically acceptable timeframe in which his symptoms could have begun or developed.

1. Petitioner Has Articulated a Sound and Reliable Medical Theory Causally Connecting Influenza Vaccine to CIDP.

To satisfy *Althen* Prong I, petitioner must present a "sound and reliable medical or scientific explanation" causally connecting the vaccine to his alleged injuries. *Knudsen,* 35 F.3d at 548.

a. Dr. Steinman's Opinions

In Dr. Steinman's opinion, petitioner suffers from CIDP caused by the influenza vaccine he received on August 19, 2013. He submits that the 2013/2014 influenza vaccine petitioner received could have triggered an immune cross-reaction to myelin and axons through molecular mimicry, leading to an immune response to antigens associated with CIDP. Pet. Ex. 25 at 16. Additionally, he submits that there are highly relevant molecular mimics with Contactin-1 and Neurofascin in the 2013/2014 flu vaccine component A/Victoria/361/2011. Pet. Ex. 25 at 9, 6.

Dr. Steinman submits that CIDP is the chronic version of GBS, with symmetrical sensory loss and chronicity – it fluctuates but goes on for a long time. Tr. 142-44; Pet. Ex. 27; Pet. Ex. 28. GBS is usually the initial diagnosis, but it becomes CIDP after a second episode. GBS "peaks and then it peters out" with or without remaining deficit, but CIDP continues with fluctuating

symptoms. Tr. 144. Steroids commonly used to treat CIDP should not be prescribed for GBS and may worsen it. Tr. 145; Pet. Ex. 27. Dr. Steinman agreed that Dr. Hopkins prescribed steroids with good response on September 17, 2013, referring to the use of steroids as an "interesting operational problem," because if the first attack was CIDP and not GBS, then prednisone was not a bad idea in retrospect. "This gets to the whole issue, people can be lumpers or splitters and say that the two diseases are very different and point to, one is acute and one is chronic, but it has to begin sometime in some patients. It begins with the first attack." Tr. 560-61. Dr. Steinman referred to NINDs, a branch of the NIH related to neurology, stating it "explicitly" states GBS and CIDP are part of the same disease entity. Tr. 560; Pet. Ex. 27.

1. The first theory involves antiganglioside antibodies through molecular mimicry.

Dr. Steinman has been studying molecular mimicry for a quarter century. The 2013/2014 influenza vaccine that petitioner received contained H1N1 influenza virus strains that can elicit antiganglioside antibodies through the mechanism of molecular mimicry, which has been associated with inflammatory neuropathy and is the same target triggered in both campylobacter jejuni infection and CIDP. Tr. 164-66, 181-82, 258; Pet. Ex. 25 at 6, 8, 16; Pet. Ex. 42;[88] Pet. Ex. 43.[89] Gangliosides are sugars and the key structures associated with the pathogenesis of inflammatory polyneuropathy. The concept of molecular mimicry involves shared structures on a virus, bacteria, or vaccine that can trigger a cross-reactive response to self: "T cells recognize foreign antigens when presented by HLA molecules of the immune system. In some people, especially those who have certain HLA types, a foreign antigen may resemble an antigen produced by the body. Such molecular mimicry provokes the T cells to attack body tissues that contain the self-antigens." Pet. Ex. 25 at 7-8.

Dr. Steinman provided a diagram of molecular mimicry, literature, and a description of the components of the flu vaccine petitioner received to show how well-received the concept of molecular mimicry is. Tr. 162-65; Pet. Ex. 25 at 5, 8; Pet. Ex. 34;[90] Pet. Ex. 77;[91] Pet. Ex. 83;[92] Pet. Ex. 84. He relied on several articles, though none addressed CIDP, to lay the groundwork for his opinion and "exemplify molecular mimicry and the components of the flu vaccine." Tr. 162-63; Pet. Ex. 34; Pet. Ex. 77; Pet. Ex. 83. He referenced a 2012 IOM report as recognizing C. jejuni infection as the exemplification of molecular mimicry and its function. Tr. 164, 167; Pet. Ex. 42.

According to Dr. Steinman, the literature supports his hypothesis in that C. jejuni is often present in poultry, and influenza vaccines are manufactured in chicken eggs. Therefore, contamination by C. jejuni of the eggs used to produce the influenza vaccine could induce GBS in

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[88] C.W. Ang et al., *Structure of Campylobacter jejuni Lipopolysaccharides Determines Antiganglioside Specificity and Clinical Features of Guillian Barre and Miller Fisher Patients*, 70 INFECTION AND IMMUNITY 1202 (2002), filed as "Pet. Ex. 42."

[89] Y. Fukami et al., *Anti-GQ1b antibody syndrome: anti-ganglioside complex reactivity determines clinical spectrum*, 23 EUROPEAN J. OF NEUROLOGY 320 (2016), filed as "Pet. Ex. 43."

[90] Syed Sohail Ahmed et al., *Antibodies to influenza nucleoprotein cross-react with human hypocretin receptor 2*, SCIENCE TRANSLATIONAL MED. (2015), http://stm.sciencemag.org/, filed as "Pet. Ex. 34."

[91] Jennifer M. Martinez-Thompson et al., *Composite Ganglioside Antibodies and Immune Treatment Response in MMN and MADSAM*, 57 MUSCLE & NERVE 1000 (2018), filed as "Pet. Ex. 77."

[92] L. Steinman and S. Ahmed, Supplementary Materials (confidential submitted supplementary materials for Ahmed et al., *supra* note 90), filed as "Pet. Ex. 83."

susceptible hosts by eliciting antiganglioside antibodies after vaccination. Pet. Ex. 41 at 2.[93] The exact pathogenesis of post-campylobacter neuropathy is unknown, but molecular mimicry between bacterial glycoconjugates and peripheral nerve gangliosides has been implicated. Cross-reactive antibodies between C. jejuni lipopolysaccharides and gangliosides have been identified in GBS and Miller Fisher Syndrome. Antiganglioside antibody reactivity against GM1, GM1b, and GaINAc-GD1a is associated with pure motor GBS, and anti-GQ1b antibody reactivity has a strong association with oculomotor symptoms and ataxia. Tr. 167-68; Pet. Ex. 42 at 1; Pet. Ex. 43. Further, cellular immunity is suspected to be implicated due to findings of macrophages and T CD4+ lymphocytes in nerve biopsies. The data support the hypothesis of an antigen-driven T cell attack against nerves, even if the target of the immune response remains unclear. Tr. 167-68; Pet. Ex. 42 at 1; Pet. Ex. 43; Pet. Ex. 51 at 1.[94]

Dr. Steinman relied on the *Nachamkin*[95] study to show that mice made antiganglioside responses, which are associated with CIDP, when the mice were injected with vaccines containing H1N1. Tr. 167, 576; Pet. Ex. 25; Pet. Ex. 41. Dr. Steinman conceded *Nachamkin* was published in 2008 and used the 1976/1977, 1991/1992, and 2004/2005 swine flu vaccines to "test the hypothesis," not the H1N1 vaccine received here. Tr. 259-260; Pet. Ex. 41. He further agreed the study concluded that more research was needed regarding influenza vaccine components eliciting antiganglioside response and the role of these antibodies, if any, in vaccine association with GBS. Tr. 260-61. He added that follow-up studies have been done, though neither he nor respondent referenced or provided them. Tr. 261, 309-310. While he submitted that the H1N1 component was integral to his theory on antigangliosides, he did not know if the H1N1 received by petitioner was the same as the H1N1 used in *Nachamkin*, which would affect the results. He was admittedly unprepared for the question and could not answer it. Tr. 310.

Dr. Steinman referred to the *Ang*[96] study as "an exquisite paper" cited in the 2012 IOM report as the best paper on molecular mimicry and how C. jejuni causes an immune response to gangliosides resulting in GBS. Tr. 266; Pet. Ex. 42. He also relied on *Fukami*,[97] which discusses the nuances of gangliosides. Pet. Ex. 43. Though vaccines were not discussed, Dr. Steinman stated the articles were submitted to show the complexity of gangliosides as fundamental to inflammatory neuropathies. Tr. 268. He stated the *Wang*[98] study shows that humans over the age of 60 produce antiganglioside antibodies after flu vaccine. Tr. 576; Pet. Ex. 95.[99]

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[93] Irving Nachamkin et al., *Anti-Ganglioside Antibody Induction by Swine (A/NJ/1976/H1N1) and Other Influenza Vaccines: Insights into Vaccine-Associated Guillain-Barre Syndrome*, 198 J. OF INFECTIOUS DISEASES 226 (2008), filed as "Pet. Ex. 41."

[94] Alexandra Richard et al., *Transcriptome Analysis of Peripheral Blood in Chronic Inflammatory Demyelinating Polyradiculoneuropathy Patients Identifies TNFR1 and TLR Pathways in the IVIg Response*, 95 MED. 1 (2016), filed as "Pet. Ex. 51."

[95] Nachamkin et al., *supra* note 93.

[96] Ang et al., *supra* note 88.

[97] Fukami et al., *supra* note 89.

[98] David J. Wang et al., *No evidence of a link between influenza vaccines and Guillain-Barre syndrome-associated antiganglioside antibodies*, 6 INFLUENZA & OTHER RESPIRATORY VIRUSES 159 (2012), filed as "Pet. Ex. 95."

[99] *Id.*

Dr. Steinman discussed a study done at the Mayo Clinic showing subtle differences between MMN, MADSAM,[100] and CIDP, all of which are chronic inflammatory neuropathies and show antiganglioside antibodies and responsiveness to IVIG. Tr. 168-69; Pet. Ex. 77. *See also* Pet. Ex. 76;[101] Pet. Ex. 78.[102] He added that section 6.2 of the 2013/2014 flu vaccine package insert contained post-marketing reports of GBS following vaccination and "more dramatically", section 5.1 contained a warning from Sanofi and the FDA of some kind of association between GBS and the vaccine. He admitted CIDP was not specifically addressed but stated he did his best to find peer-reviewed literature to show that the flu vaccine can trigger an inflammatory neuropathy. Tr. 161, 577; Pet. Ex. 84 at 11.

Initially, Dr. Steinman discounted Type 2 diabetes as a cause of CIDP, relying on a case study of an insulin-dependent patient with Type 2 diabetes and neuropathy who developed CIDP following campylobacter infection. The campylobacter infection, not the Type 2 diabetes, was determined to be the cause of the inflammatory demyelinating neuropathy through molecular mimicry. Pet. Ex. 25; Tr. 155-57; Pet. Ex. 36.

2. Dr. Steinman's other theory involves Contactin I and Neurofascin.

Dr. Steinman presented another theory that involves "highly relevant molecular mimics with contactin-1 and neurofascin [are] contained in the flu vaccine component A/Victoria/362/2011." Pet. Ex. 25 at 6, 9. Unlike gangliosides, Contactin-1 and Neurofascin are proteins with amino acid homology that "can cause neuroinflammatory disease with clinical paralysis, when such a molecular mimic is injected into an experimental animal." Pet. Ex. 25 at 10; Tr. 170; Pet. Ex. 78. These proteins are located on the peripheral nerve where the electrical current jumps over the axons present on the myelin sheath. The antibodies directed against Contactin-1 and Neurofascin are most likely the IgG4 isotype and are associated with aggressive symptom onset, sensory ataxia, tremor, and poor response to IVIG treatment. Pet. Ex. 45 at 5.[103] The reasons that Contactin-1 affects motor neurons are unknown. Tr. 170-71; Pet. Ex. 46 at 5, 8;[104] Pet. Ex. 47 at 9.[105]

Dr. Steinman relied on *Devaux*[106] to show that of 533 CIDP patients studied, 38 or 7 percent were found to have anti-neurofascin 155 IgG4 antibodies, which was impressive because it is very rare. Pet. Ex. 43; Pet. Ex. 45 at 5, 8. The group with antibodies and CIDP were younger at onset and had ataxia, tremors, CNS demyelination, and poor response to IVIG. Tr. 275; Pet. Ex. 43 at 1; Pet. Ex. 45 at 5, 8; *see also* Pet. Ex 46 at 1-2.

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[100] MMN stands for multifocal motor neuropathy; MADSAN stands for multifocal acquired demyelinating sensor and motor neuropathy. *See* Pet. Ex. 77 at 1.

[101] Juliane Klehmet et al., *Analysis of anti-ganglioside antibodies by a line immunoassay in patients with chronic-inflammatory demyelinating polyneuropathies (CIDP)*, 56 CLIN. CHEM. LAB. MED. 919 (2018), filed as "Pet. Ex. 76."

[102] Luis Querol et al., *Antibodies against peripheral nerve antigens in chronic inflammatory demyelinating polyradiculoneuropathy*, 7 SCI. REP. 1 (2017), filed as "Pet. Ex. 78."

[103] Jérôme J. Devaux et al., *Neurofascin-155 IgG4 in chronic inflammatory demyelinating polyneuropathy*, 86 NEUROLOGY 800 (2016), filed as "Pet. Ex. 45."

[104] Yumako Miura et al., *Contactin I IgG4 associates to chronic inflammatory demyelinating polyneuropathy with sensory ataxia*, 138 BRAIN 1484 (2015), filed as "Pet. Ex. 46."

[105] Constance Manso et al., *Contactin-I IgG4 antibodies cause paranode dismantling and conduction defects*, 139 BRAIN 1700 (2016), filed as "Pet. Ex. 47."

[106] Devaux et al., *supra* note 103.

Dr. Steinman conducted BLAST searches comparing A/Victoria/361/2011 in the subject vaccine to Contactin-1 and Neurofascin to identify sequences of homology between the two proteins and the subject vaccine. Tr. 258, 273. The BLAST search for Neurofascin found 7 out of 9 identical amino acids, while the BLAST search for Contactin-1 found 5 out of 6 identical amino acids in one area and 5 out of 10 identical amino acids in another. Tr. 172-73. He stated that "[t]his degree of homology can cause neuroinflammatory disease with clinical paralysis, when such a molecular mimic is injected into an experimental animal," citing to studies in his laboratory showing 5 out of 12 amino acids and 4 out of 11 nonconsecutive amino acids as sufficient to trigger experimental encephalomyelitis (EAE) in mice. Pet. Ex. 25 at 11-12, 14-15. The results from Dr. Steinman's lab studies imply the components of the 2013/2014 influenza vaccine can trigger inflammatory neuropathy, or in his study, EAE, via an immune response to gangliosides, Contactin-1, or Neurofascin, each associated with CIDP. Pet. Ex. 25 at 15; Pet. Ex. 74 at 1, supp. ref. 2,[107] supp. ref. 3.[108]

He agreed that the mouse studies showing that 5 out of 12 identical amino acids were sufficient to cause paralysis did not study CIDP, but rather whether something in a virus or vaccine that had homology with something in a protein would cause paralysis in the animal when the system was "revved up" with an adjuvant. Tr. 172-73, 269-270. However, the finding of 5 out of 12 amino acids needed to cause paralysis was "striking." He agreed the study used Freund adjuvant,[109] a potent adjuvant, but that was so a million mice did not have to be killed. Tr. 270. Dr. Steinman stated, "people criticize the study," but it was peer reviewed, which is a rigorous process by people who don't like him, so "you can take or leave it" and "[i]f you don't like my analogy with EAE or how much identity you need, if you don't like the fact that it's a peer-reviewed publication, I'm not here to elevate beyond what it is." Tr. 173-75, 271.

Dr. Steinman disagreed that two proteins could have similar segment homology by random chance rather than clinical significance. Tr. 272-73.

Dr. Steinman conceded there is no epidemiologic evidence that flu vaccine can cause CIDP but relies on his service to the Department of Justice in the 1980s and his testimony about whether the swine flu vaccine triggered GBS, though it is unclear if the swine flu vaccine and GBS is an adequate surrogate for the 2013/2014 influenza vaccine and GBS/CIDP. Tr. 154-55; Pet. Ex. 25 at 16.

Dr. Steinman remarked that Dr. Chaudhry did not challenge the molecular mimicry theory other than to say petitioner did not have the antibodies or clinical presentation for anti-Neurofascin or Contactin-1, which includes sensory ataxia and tremors. Dr. Steinman conceded the literature he relied on for his theory of Contactin-1 and Neurofascin as a cause of CIDP did not look at post-vaccination, but the best he could say is it would be the same. Tr. 182.

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[107] Martinez-Thompson et al., *supra* note 91.
[108] Querol et al., *supra* note 102.
[109] Freund adjuvant is a water-in-oil emulsion incorporating antigen, in aqueous phase into light weight paraffin oil with the aid of emulsifying agent. "On injection, this mixture…induces strong persistent antibody formation." *Dorland's* 32.

b. Dr. Chaudhry's opinion

Dr. Chaudhry submits that the concept of H1N1 influenza vaccine eliciting antiganglioside antibodies is controversial, but more importantly, antiganglioside antibodies have not been generally associated with CIDP. Resp. Ex. A at 11; Resp. Ex. D at 11. Further, Dr. Chaudhry disagrees that GBS and CIDP are a continuum of the same disease, adding that Dr. Steinman relied on a definition of CIDP from the NIH website that is written for laypeople and does not provide the pathogenesis, which is different for GBS and CIDP as demonstrated by responses to treatment. However, Dr. Chaudhry deferred to Dr. Steinman's expertise on molecular mimicry conceding he may be correct, but today's science does not support CIDP due to molecular mimicry. Tr. 431-32.

Dr. Chaudhry explained that not all forms of GBS or CIDP are the same and not all have molecular mimicry. Acute motor axonal neuropathy ("AMAN") and Miller Fisher variant are the only forms of GBS associated with molecular mimicry. Tr. 430. CIDP has several forms, but a "triggering agent has not been found except for in rare cases of CIDP associated with melanoma in which the tumor cells share carbohydrates, epitopes and Schwann cells." That is the only molecular mimicry cause of CIDP known to date. Tr. 430-31, 433; Resp. Ex. A, Tab 2 at 2.[110]

Dr. Chaudhry commented on several papers submitted by Dr. Steinman discussing MMN, MADSAM, and CIDP, stating that they are different diseases and only 10 percent have antibodies. The gangliosides are different kinds of IgM and IgG, and IgM GN1 gangliosides are found in 60 to 70 percent of multifocal motor neuropathy patients, but they have not been noted as pathogenic. Patients have antibodies to gangliosides in a lot of different conditions, but whether they produce a response is not known for CIDP and their significance is currently unknown. Tr. 434.

In the *Nachamkin*[111] study, Dr. Chaudhry pointed out that the mice developed antibodies after receiving double or triple the dose of injection and yet did not become clinically symptomatic or develop GBS. Yuki, who discovered the molecular mimicry hypothesis for GBS in AMAN resulting from c. jejuni infection, was approached by the WHO to conduct a study over concern for vaccine safety. He did not find any of the flu vaccines studied to have ganglioside antibody response in men or in mice. Further, CIDP is not known to be caused by ganglioside antibodies, and more importantly, Dr. Chaudhry stated petitioner did not have CIDP, so the hypothesis for molecular mimicry is irrelevant. Tr. 438-39.

Dr. Chaudhry agreed Contactin-1 and Neurofascin can affect the axon and cause axonal damage, but each of the only nine patients known to have had Contactin-1 and Neurofascin antibody-induced CIDP had very abnormal EMGs. Tr. 355-56. The younger patients had sensory ataxia with tremor, clear EMG findings, and a lack of response to IVIG. Summarily, the literature addressing Contactin-1 and Neurofascin antibodies reported in CIDP showed objective evidence of marked demyelination on EMG and a lack of response to IVIG. Tr. 435-36; Resp. Ex. A at 11. Further, the literature Dr. Steinman relied on for this theory does not discuss CIDP, it discusses EAE. Resp. Ex. A at 11.

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[110] Jean-Michel Vallat, *Chronic inflammatory demyelinating polyradiculopathy: diagnostic and therapeutic challenges for a treatable condition*, 9 LANCET NEUROL. 402 (2010), filed as "Resp. Ex. A, Tab 2."
[111] Nachamkin et al., *supra* note 93.

Dr. Chaudhry deferred to Dr. Steinman's knowledge of immunology and the use of BLAST search similarities and hypotheses. However, there are no Contactin-1 or Neurofascin antibodies causing CIDP in his own patients with CIDP, and further, CIDP presents differently than the onset of GBS two to three weeks after infection. He asks all his patients if they had vaccines or an infection and the CIDP patients do not provide a history of either. Tr. 435-37, 456-58.

Dr. Chaudhry agreed there were issues years ago with GBS following the swine flu vaccine, but he has only seen one or two patients with GBS after flu vaccine in the past 30 years. The cause and effect are still up in the air, but he accepts GBS after flu vaccine as a table injury. Tr. 459-460. However, the same cannot be said for CIDP, as there is no data to support vaccines causing CIDP. Tr. 460-62.

c. Analysis of Prong I

Dr. Steinman opined that CIDP is the chronic form of GBS, and petitioner developed GBS and/or CIDP from the flu vaccine. Pet. Ex. 25; Pet. Ex. 74. His theories include molecular mimicry involving antigangliosides and highly relevant molecular mimics with Contactin-1 and Neurofascin that existed in the 2013/2014 flu vaccine component A/Victoria/361/2011, with several homologies "that can cause neuroinflammatory disease with clinical paralysis, when such a molecular mimic is injected into an experimental animal." Pet. Ex. 25 at 6, 9, 10. He concluded that components of the 2013/2014 influenza vaccine can trigger inflammatory neuropathy via an immune response to gangliosides, Contactin-1, or Neurofascin, as each antigen is associated with CIDP. *Id*. at 15.

Dr. Chaudhry did not disagree with the theory of molecular mimicry per se, deferring to Dr. Steinman's expertise, but he remarked that Contactin-1 and Neurofascin is very rare and has only been detected in nine patients with CIDP who had distinct symptoms that do not exist in this case. He disagreed that CIDP is the chronic form of GBS because the two diseases have different pathogenesis and treatment, and he disagreed that there is support for molecular mimicry occurring in CIDP, except with melanoma. Most importantly, he disagreed that GBS or CIDP was the correct diagnosis in this case.

The experts are credited with having extensive knowledge and experience with these diseases and spending significant time discussing whether a causal link exists between the flu vaccine and CIDP via theories of molecular mimicry or Contactin-1 and Neurofascin. Prior cases in the program have addressed whether GBS and CIDP are distinct diseases or a continuum of the same disease, referring to the two as "related" peripheral neuropathies with a number of overlapping symptoms that "may" share a common pathogenesis. *See Strong v. Sec'y of Health & Human Servs*., No. 15-1108V, 2018 WL 1125666 (Fed. Cl. Spec. Mstr. Jan. 12, 2018) (referencing the "large and persuasive" body of evidence reliably connecting flu vaccine and GBS and noting the common symptoms and pathogenesis of CIDP and GBS); *Daily v. Sec'y of Health & Human Servs*., 2011 WL 2174535 (Fed. Cl. Spec. Mstr. May 11, 2011) (finding that a theory of CIDP caused by molecular mimicry is plausible based on the premises that vaccination can cause GBS, molecular mimicry can cause GBS, and there is a biological basis for similarity between GBS and CIDP). In that regard, there is agreement that petitioner can satisfy *Althen* Prong I in a case like this by relying on the theory of molecular mimicry. This decision therefore does not reach any

conclusions about Dr. Steinman's theory involving Contactin-1 and/or Neurofascin, as there is nothing in the record to suggest the presence of either Contactin-1 or Neurofascin in petitioner. Thus, petitioner has established a medical theory causally linking the flu vaccine to GBS and CIDP by molecular mimicry, satisfying Prong I.

2. Petitioner Has Demonstrated a Logical Sequence of Cause and Effect That the Receipt of the Influenza Vaccine Triggered an Immune-Mediated Inflammatory Reaction that Contributed to His Already Compromised Neurological Condition from Various Comorbidities.

Having already determined Prong I and in order to determine Prong II, petitioner's diagnosis must be addressed. As a threshold matter, petitioner must first establish that he actually suffered the injury alleged in the petition. *See Broekelschen v. HHS*, 618 F.3d 1339, 1346 (Fed. Cir. 2010). As the Federal Circuit has made clear, "the statute places the burden on petitioner to make a showing of at least one defined and recognized injury." *Lombardi v. HHS*, 656 F.3d 1343, 1353 (Fed. Cir. 2011) (affirming a special master's decision to dismiss a petition when the petitioner could not establish that she had any of the three diagnoses alleged). "The function of a special master is not to 'diagnose' vaccine-related injuries, but instead to determine based on the record evidence as a whole and the totality of the case, whether it has been shown by a preponderance of the evidence that a vaccine caused [petitioner's] injury." *Lombardi*, 656 F.3d at 1352-53 (internal citation omitted). Thus, where "the existence and nature of the injury itself is in dispute, it is the special master's duty to *first determine* which injury is best supported" by the evidence. *Id*. at 1352 (citing *Broekelschen*, 618 F.3d at 1345) (emphasis added).

a. Overview of Guilain Barre Syndrome, Chronic Inflammatory Demyelinating Polyneuropathy, Diabetes Mellitus, and Diabetes Mellitus with Neuropathy

Guillain-Barre Syndrome ("GBS") is a disorder in which the body's immune system starts to destroy the myelin sheath that surrounds the axons of many peripheral nerves or the axons themselves. Pet. Ex. 27 at 1.[112] GBS presents with varying degrees of weakness or tingling sensations in the legs and ascends symmetrically to the arms and upper body. GBS can affect anyone at any age and usually occurs a few days or weeks after respiratory or gastrointestinal viral infection. GBS can also be triggered by surgery, and vaccinations may increase the risk of GBS in rare instances. *Id*.[113] There are varying degrees of progression of the disease, and it can be life

[112] *Guillain-Barre Syndrome Fact Sheet*, NAT'L INST. OF NEUROLOGICAL DISORDERS AND STROKE, http://ninds nih.gov (Mar. 30, 2017, 1:07 PM), filed as "Pet. Ex. 27."

[113] In March 2017, GBS was added to the table of vaccine injuries associated with the influenza vaccine, occurring within 3-42 days following vaccination. - the criteria for a Table GBS injury in the Act's qualifications and aids to interpretation ("QAI"), set forth at 42 C.F.R. § 100.3(c)(15). The QAI specify that: GBS is an acute monophasic peripheral neuropathy that encompasses a spectrum of four clinicopathological subtypes . . . [T]he interval between the first appearance of symptoms and the nadir of weakness is between 12 hours and 28 days. This is followed in all subtypes by a clinical plateau with stabilization at the nadir of symptoms, or subsequent improvement without significant relapse. Treatment related fluctuations in all subtypes of GBS can occur within 9 weeks of GBS symptom onset and recurrence of symptoms after this time-frame would not be consistent with GBS. 42 C.F.R. § 100.3(c)(15)(i). To "qualify as any subtype of GBS, there must not be a more likely alternative diagnosis for the weakness." 42 C.F.R. § 100.3(c)(15)(v). In particular, the "[e]xclusionary criteria for the diagnosis of all subtypes of GBS include the ultimate diagnosis of any of the [listed] conditions," and the list is not exhaustive. *See* 42 C.F.R. § 100.3(c)(15)(vi); *see also* 42 U.S.C. § 300aa-13(a)(1).

threatening. However, most individuals have a good recovery, although some have a certain degree of weakness. *Id*. Oral steroids and intravenous methylprednisolone are not beneficial in treating GBS. Resp. Ex. C, Tab 1 at 8.[114]

Chronic inflammatory demyelinating polyneuropathy ("CIDP"), sometimes called chronic relapsing polyneuropathy, is an immune-related, idiopathic, clinically heterogeneous disorder which causes damage to the myelin sheath of the peripheral nerves. Pet. Ex. 36 at 1[115]; Pet. Ex. 28 at 1.[116] It is characterized by progressive weakness and areflexia, with features of demyelination including prolonged distal and F-wave latencies, reduced conduction velocity, conduction block and temporal dispersion on electrophysiological testing, and inflammation and demyelination/remyelination on nerve biopsy. Resp. Ex. A at 9. The diagnostic criteria for CIDP are sufficiently broad to include all patients who could benefit from immunomodulatory treatment. Pet. Ex. 68 at 1.[117] Typical (sensorimotor, symmetrical, predominantly proximal weakness) and atypical (predominantly distal weakness, focal presentations, pure sensory, pure motor, and pure ataxia) variants are accepted to lie within the CIDP spectrum. Pet. Ex. 68 at 1; Pet. Ex. 37 at 1.[118] Clinical criteria include motor or sensory dysfunction in more than one limb for more than two months with hyporeflexia and characteristic CSF and electrodiagnostic test results. Pet. Ex. 38 at 1.[119] Some CIDP subtypes exhibit differences in disease progression (relapsing or progressive), associated clinical features (cranial involvement), concomitant disease (diabetes mellitus), and paraclinical features (IgG or IgA monoclonal gammopathy) that further broaden the spectrum of disease. Pet. Ex. 68 at 1.

CIDP can occur at any age and presents with symptoms of tingling or numbness starting in the toes and fingers, weakness of the arms and legs, loss of deep tendon reflexes, fatigue, and abnormal sensations. Pet. Ex. 28 at 1. A good response to intravenous immunoglobulin (IVIg) and plasma exchange suggests a pathogenetic contribution of humoral factors, including autoantibodies. Pet. Ex. 68 at 2. Some patients with CIDP have a spontaneous recovery, while many have bouts of symptoms with partial recovery in between relapses. Some individuals are left with residual numbness or weakness. *Id*. at 1.

A diagnosis of CIDP is based on clinical features, neurological examination, and electrodiagnostic criteria including EMG and NCS, which are necessary to confirm the diagnosis. Laboratory testing, elevated CSF protein levels with a leukocyte count of less than 10 cells/mm, MRIs of the lumbosacral and cervical nerve roots or brachial or lumbosacral plexuses, nerve

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[114] Hugh J. Willison et al., *Guillain-Barre syndrome*, 388 LANCET 717 (2016), filed as "Resp. Ex. C, Tab 1."

[115] Yusuf A. Rajabally et al., *Chronic inflammatory demyelinating polyneuropathy after Campylobacter jejuni infection mimicking vasculitic mononeuritis multiplex in a diabetic*, J. PERIPHERAL NERVOUS SYS. 98 (2004), filed as "Pet. Ex. 36."

[116] *Chronic Inflammatory Demyelinating Polyneuropathy (CIDP) Information Page*, NAT'L INST. OF NEUROLOGICAL DISORDERS AND STROKE, http://ninds nih.gov (Mar. 30, 2017, 1:27 PM), filed as "Pet. Ex. 28."

[117] Luis Querol et al., *Autoantibodies in chronic inflammatory neuropathies: diagnostic and therapeutic implications*, 13 NATURE REVS. NEUROLOGY 533 (2017), filed as "Pet. Ex. 68."

[118] A. Chiò et al., *Comorbidity between CIDP and diabetes mellitus: only a matter of chance?*, 16 EUROPEAN J. OF NEUROLOGY 752 (2009), filed as "Pet. Ex. 37."

[119] J.M. Brostoff et al., *Post-influenza vaccine chronic inflammatory demyelinating polyneuropathy*, 37 AGE AND AGING 229 (2007), filed as "Pet. Ex. 38."

biopsy, and clinical improvement after immunomodulatory treatment can help rule out other causes for neuropathy and support the diagnosis. Resp. Ex. A, Tab 5 at 2-3.[120]

The 2010 European Federation of Neurological Societies/Peripheral Nerve Society (EFNS/PNS) guidelines are "globally accepted for both clinical and research purposes due to their high sensitivity and specificity for CIDP." Resp. Ex. A, Tab 5 at 3. "The EFNS/PNS defines CIDP as 'definite,' 'probable,' or 'possible,' based on motor distal latency prolongation, reduction of motor conduction velocity, prolongation or absence of F-waves, motor conduction block, and distal compounds muscle action potential (CMAP) duration." *Id*. Testing of multiple limbs is more sensitive for atypical CIDP. *Id*.

Both GBS and CIDP are considered immune-mediated polyneuropathies associated with a variable clinical course and outcome. "GBS patients usually reach their maximum disability within 4 weeks of onset compared to at least 2 months for CIDP." Pet. Ex. 39 at 1.[121] A June 2008 study by questionnaire conducted on 461 members of the Dutch society of neuromuscular disorders with GBS and CIDP focused on four areas: preceding vaccinations within 8 weeks of onset of GBS or CIDP, family members with GBS or CIDP, occurrence of common auto-immune disease, and persistent symptoms at a variable time point after the diagnosis. *Id.* at 3. Of the 323 questionnaires returned, 23 GBS and 8 CIDP patients reported onset within 8 weeks of vaccination, most often the flu vaccination. None of the 106 GBS patients reported recurrence after subsequent flu vaccine. Of 24 patients with CIDP after the flu vaccine, 5 reported an increase in symptoms after one or more vaccinations. *Id*. There were two patients with both GBS and CIDP. "Although the combination of having both GBS and CIDP could be by chance, these patients support the hypothesis that GBS and CIDP may constitute a clinical continuum or that there are common host factors which influence susceptibility to these disorders." *Id.* at 4. The study showed that subsequent flu vaccination was safe for those who had suffered GBS or CIDP following vaccination, but a host of biases associated with the study by questionnaire were noted. *Id*. at 5-6.

Petitioner submitted a case study of a 74-year-old who developed progressive right-sided paresthesia and weakness with dysarthria, ascending weakness of upper and lower limbs, and exertional dyspnea two days after receipt of a flu vaccine. He had a prior history of coronary artery bypass grafting, gout, and chronic renal impairment. Pet. Ex. 38 at 1. At this time, CIDP after flu vaccine had not been previously reported. Onset was rapid compared to the latent period of approximately two weeks between vaccine and GBS, and the initial presentation was unusual, with facial numbness, dysarthria with prominent symptoms of dyspnea, and unresponsiveness after the second immunoglobulin course. Pet. Ex. 38 at 1-2. The study noted:

> Viruses and viral vaccines have been proposed as putative triggers in the pathogenesis of autoimmune disease with postulated mechanisms including antigen mimicry, triggering self-reactive T-cell clones, and cytokine upregulation that may induce aberrant MHC class II expression. Whilst autoimmune neurological sequelae of influenza vaccination have

[120] Vera Bril et al., *The dilemma of diabetes in chronic inflammatory demyelinating polyneuropathy*, 30 J. DIABETES COMPLICATIONS 1401 (2016), filed as "Resp. Ex. A, Tab 5."
[121] Krista Kuitwaard et al., *Recurrences, vaccinations and long-term symptoms in GBS and CIDP*, 14 J. PERIPHERAL NERVOUS SYS. 310 (2009), filed as "Pet. Ex. 39."

> been described, the development of CIDP after influenza vaccination has not been previously reported.

Pet. Ex. 38 at 1. The study reached no conclusions regarding the correlation of the gentleman's flu vaccine and onset of atypical CIDP other than to say that "[r]arely, individual patients may develop certain restricted patterns of autoimmune neurological damage and physicians need to be aware of novel presentations." *Id*. at 2.

Diabetes mellitus ("DM") affects about 9.3 percent of the general population in the United States, but 25.9 percent of persons 65 and over. Type 2 DM accounts for more than 90 percent of the cases and results in insulin resistance. Prevalence of Type 2 DM increases with age, elevated body mass, and family history. Type 2 DM has a gradual onset and early symptoms may go unnoticed. Resp. Ex. A, Tab 5 at 3. Neuropathy is somewhat common in patients with DM: "[w]hile it is estimated that 50% of patients with DM have some form of neuropathy, more than 80% of these cases are diabetic peripheral neuropathy (DPN) – a length dependent, sensory more than motor, axonal neuropathy." Resp. Ex. A, Tab 5 at 4; Resp. Ex. D, Tab 3 at 7.[122] Early diabetic neuropathy manifests with distal loss of sensation in the feet and/or loss of deep tendon reflexes in the ankles. The risk of developing diabetic neuropathy increases with duration of DM and glycemic control. Resp. Ex. A, Tab 5 at 4.

CIDP is reported more frequently in patients with Type 2 DM and diagnosing CIDP in these patients is more challenging due to superimposed axonal damage that can obscure EMG findings. Further, DM can also cause elevated protein in CSF, which is part of diagnostic criteria for CIDP. Resp. Ex. A, Tab 5 at 2, 4; Resp. Ex. D, Tab 3 at 8. According to a health insurance administration claims database study, the prevalence of CIDP in the non-diabetic population is 6 per 100,000 people, while the prevalence of CIDP in the DM population is 54 per 100,000 people. In addition to the concomitant axonal damage, the increased prevalence of both CIDP and DM in the over 50 population also creates controversy in the association of the two. While patients with CIDP and DM are both responsive to immunological treatment, more definite testing is needed to distinguish the two conditions. Resp. Ex. A, Tab 5 at 7, 9. However, response to "immunoglobulin therapy, although non-specific, indicates an immune basis to the neuropathy." Pet. Ex. 36 at 4.

While the etiology of CIDP is unknown, "more than one-third of cases are associated with other disorders," including DM. Pet. Ex. 37 at 1. A 2001 article submitted addressing the association between CIDP and DM suggested it "seems to be just coincidental", but later studies suggest otherwise. *Id.* at 2. The 2001 article concluded that epidemiological findings do not support a pathogenetic correlation between DM and CIPD. However, when a patient with DM has symptoms of peripheral neuropathy, "a thorough EP [electrophysiological] examination is required to search for signs of demyelination, meeting the diagnostic criteria for CIDP, as some patients with CIDP may have response to therapy, differently from those with the classic axonal diabetic neuropathy." *Id*. at 3.

In 2004, a study noted, "Recent reports suggest that CIDP could be more frequent in diabetics." Pet. Ex. 36 at 1. Superimposed on an axonal polyneuropathy, CIDP probably occurs

[122] Gérard Said, *Diabetic Neuropathy*, in Handbook of Clinical Neurology 579-89 (G. Said & C. Krarup eds., 2013), filed as "Resp. Ex. D, Tab 3."

more frequently in diabetics, which itself results in various types of neuropathies. *Id*. Though the exact incidence of CIDP in diabetics remains uncertain, it is probably much higher than in the general population. *Id*. at 5. Only 60-70 percent of sural nerve biopsies in CIDP show typical signs of demyelination, and though rare, diabetic neuropathies can fulfill electrophysiological criteria for CIDP. *Id* at 4. The severity of symptoms, marked distal weakness, and imbalance could represent clinical indicators of CIDP superimposed on a diabetic polyneuropathy, though the diagnosis remains difficult in practice. *Id.* at 5.

b. Dr. Steinman's Opinion

Dr. Steinman adamantly argues that petitioner has CIDP because of the "repeated use of the word 'CIDP' as a diagnosis" throughout the medical records and the administration of "very expensive medicines" like IVIG, which have serious side effects. Tr. 150. Dr. Steinman reviewed the record "extensively," and concluded "a lot" of doctors, specialists, and institutions diagnosed petitioner with CIDP. Tr. 140-41. He stated petitioner's treating physicians couched their diagnosis in terms of "possible or probable CIDP" rather than just "CIDP" because they were not writing an opinion for the Vaccine Program. However, he is, and petitioner has CIDP. Pet. Ex. 61 at 6. "I think the influenza vaccine caused the CIDP. And secondarily, it could have aggravated an underlying neuropathy." Tr. 233.

Dr. Steinman addressed the other diagnoses contained in the medical records and explained why, in his opinion, these diagnoses did not apply to petitioner. Infective polymyositis was most likely referring to acute inflammatory demyelinating polyneuropathy ("AIDP") or GBS. Tr. 146; Pet. Ex. 3 at 97. Sarcoid is an inflammatory attack, and if petitioner's treaters really thought he had sarcoid, they would have done a biopsy to confirm it. According to Dr. Steinman, there is insufficient evidence in the record to support sarcoid, and he "is constrained by what's in the record." Tr. 546-47. Similarly, though imaging would be helpful, there was no evidence of long motor track central nervous system involvement suggestive of myelopathy, but the stimulator in petitioner's back obviated the use of an MRI. Dr. Steinman stated he would have done either an MRI or CT if petitioner was his patient. Tr. 554-55.

Dr. Steinman described proximal myopathy as a disorder of the big muscles of the shoulders and hips, rather than the small muscles of the hand and feet. Tr. 145. Statins, a class of medication used to reduce cholesterol, can cause myopathy, but if petitioner's treaters really thought statins were the culprit, they would have "nailed that down." Tr. 548. He attributed petitioner's normal EMG results to having received steroids and IVIG prior to testing, but "short of doing a biopsy, which again has limitations because you might not biopsy the right muscle or region of the muscle", there was no support in the record for myopathy. Tr. 554. Myositis was unlikely because petitioner's blood work, particularly CPK, which measures muscle damage and sedimentation rate, was normal. Tr. 147. Similarly, petitioner's lab result for acetylcholine, the immunological receptor found in myasthenia gravis, was normal and the classic tests such as single fiber jitter recordings were not done. Dr. Steinman further stated IVIG is used to treat myasthenia gravis, so petitioner's lab values may have normalized from treatment. Tr. 556, 566. He tests his

patients several times when he suspects myasthenia gravis[123] and does not believe petitioner has myasthenia gravis. He stated petitioner's doctors never suggested single fiber and repetitive stimulation studies during his EMG testing, so they were not "enthusiastic" about that diagnosis either.[124] Tr. 566. According to Dr. Steinman, though various alternate diagnoses were raised, only a couple of tests were pursued, and those diagnoses were dismissed. Tr. 556, 566.

In arriving at the conclusion that petitioner's diagnosis is CIDP, Dr. Steinman placed "high weight" on petitioner's "board-certified" treating doctors, which to him means "a really seriously high standard of knowledge." Tr. 149.[125]

1. Petitioner's medical history

Dr. Steinman testified in detail that petitioner's medical history following receipt of the flu vaccine is supportive of CIDP. He stated petitioner's initial presentation on September 29, 2013 with grip strength weakness, pain in his palms, and upper and lower proximal muscle weakness was "as clear as you can be." Tr. 150-51. But Dr. Steinman noted that petitioner had trace reflexes in his knees, ankles, and triceps, and did not have upgoing toes, which is indicative of something outside the peripheral nervous system. Tr. 151. Dr. Steinman stated the nerves from the spinal cord to the ankles "are the first to go" in GBS and CIDP, admitting this also occurs with neuropathic conditions like diabetes. Tr. 566-67. Dr. Steinman claimed nothing at that visit makes or breaks the diagnosis of CIDP; the overall picture, history, and exam led several doctors to conclude a diagnosis of CIDP despite the imperfections of the diagnosis. Tr. 153.

According to Dr. Steinman, petitioner was diagnosed with CIDP after he suffered a recurrence of weakness and numbness with sensory ataxia, which is an inability to feel your arms and legs in space that causes difficulty in balance and gait. Tr. 177-78. He referenced petitioner's testimony about the car show when the security guard mistook his behavior for being drunk as indicative of sensory loss and motor weakness due to peripheral neuropathy. Tr. 178. Dr. Steinman agreed that sensory ataxia is also a symptom of diabetic neuropathy. Tr. 180-81.

Acknowledging that petitioner had Type 2 diabetes with neuropathy, Dr. Steinman initially argued that the flu vaccine was the only factor in his CIDP based on his activity level prior to the vaccine and his "downhill disability" following the vaccine. Tr. 155. He agreed diabetic neuropathy can escalate quickly but stated the picture here was more consistent with the vaccine temporally causing CIDP with "a catastrophic decline that we saw after the vaccine." Tr. 218-19, 267. Dr. Steinman relied on a study conducted in two regions of Italy and reported in the European Journal of Neurology that found no support for a pathological correlation between CIDP and DM. Pet. Ex. 25; Tr. 155-56; Pet. Ex. 37. He acknowledged the literature submitted by respondent that

[123] The characteristics of myasthenia gravis "include muscle fatigue and exhaustion that fluctuates in severity, without sensory disturbance or atrophy." It especially affects muscles of the eyes, face, lips, tongue, throat, and neck. *Dorland's* 1197.

[124] Dr. Knox conducted single fiber testing twice during EMG testing. *See* Pet. Ex. 5.

[125] Dr. Steinman placed great value on petitioner's treaters' board certifications in neurology with repeated references to a higher level of expertise, unless it was a treater who disagreed with the CIDP diagnosis. Notably while board certified in neurology, Dr. Steinman is not board certified in immunology though he has been recognized repetitively as an expert in immunology in the program.

showed a relationship between DM and CIDP, but stated the causative relationship was an open-ended question and the study he submitted points away from any relationship. Tr. 156; Pet. Ex. 37.

Rebuking the idea that petitioner's Type 2 diabetes with peripheral neuropathy contributed to petitioner's illness and/or condition, Dr. Steinman stated that the treatment for diabetic neuropathy includes diabetes control and pain medication like Gabapentin, which petitioner was taking prior to his vaccination. Tr. 158. IVIG is the medication of choice for CIDP and is far too expensive with too many potential complications to use to treat diabetic neuropathy when insulin is cheaper. Tr. 160, 550-51, 563. However, Dr. Steinman conceded that IVIG is approved for treating myasthenia gravis, polymyositis, and GBS, as well as CIDP. Prednisone would be contraindicated for someone with diabetes, but is used to treat myositis, myopathies, CIDP, and central nervous system inflammation. Tr. 550-51, 563. He added that petitioner was also treated with Cellcept, which is not used to treat diabetes, so petitioner's treaters therefore did not consider his symptoms to be diabetes related. Tr. 160, 550-51. He disagreed that petitioner was prescribed IVIG for the purpose of covering all bases of the various diagnoses being entertained; stating IVIG was prescribed because he has CIDP. Tr. 550-51, 563. When asked if petitioner's IVIG was discontinued due the diagnosis being questioned, after much posturing he responded, "I can't – I don't know"; he found nothing explicit in the chart explaining why IVIG was discontinued. Tr. 562-64.

Dr. Steinman stated that Drs. Hopkins and Mahmood documented "flu shot reaction" as affirmative proof of the relationship between the vaccine and CIDP, though he claimed this did not influence his opinion. Tr. 183-84. He then stated while Drs. Hopkins and Mahmood mentioned the flu vaccine, a doctor's job is treatment, not cause. Tr. 210. He agreed that Dr. Hopkins's September 17, 2013 office record only noted "flu shot reaction" with no mention of GBS or CIDP but pointed to the Dr. Hopkin's record in November 2013 as referring to a GBS episode. Tr. 294-95, 297.

Dr. Steinman remarked that five treating physicians thought petitioner had CIDP and prescribed IVIG and immunosuppressants even though the EMG results were not "good enough." Tr. 192-93; Pet. Ex. 55. In his 2017 report, he referred to four board-certified neurologists who diagnosed petitioner with CIDP: Drs. Mahmood, Seminer, Hu, and Knox. At hearing, he added Dr. Brown. Tr. 280; Pet. Ex. 55. He then added Dr. Hopkins, even though he is not a neurologist, stating he included GBS in his November 2013 record. Tr. 297; Pet. Ex. 2 at 13. Dr. Steinman agreed the doctors' opinions were in a state of flux, but petitioner was ultimately treated for CIDP. Tr. 279.

Dr. Steinman noted that Dr. Mahmood's initial diagnosis on September 29, 2013 was GBS. Tr. 286; Pet. Ex. 55 at 2. When he saw petitioner during his second hospitalization, he still referred to petitioner's last presentation as GBS and prescribed IVIG. Dr. Steinman agreed myopathy was included in Dr. Mahmood's differential diagnosis and that he wrote post-vaccine GBS would not explain petitioner's muscle tenderness and weakness. Tr. 286, 298-99; Pet. Ex. 3 at 69.

Dr. Steinman agreed that Dr. Seminer's record of January 27, 2014 documented the first EMG/NCS study as nondiagnostic, with an incidental finding of carpal tunnel and no convincing evidence of diffused peripheral neuropathy but pointed out that Dr. Seminer wrote in the record

several pages later that the EMG was slightly abnormal, and petitioner had CIDP with good response to IVIG. Tr. 93-194, 279, 286; Pet. Ex. 3 at 105, 113. Dr. Steinman explained that, in the real world, doctors change their minds on diagnoses all the time. Tr. 195. He also noted that Dr. Seminer wrote to Dr. Hopkins advising that Dr. Mahmood thought petitioner had CIDP. Tr. 312; Pet. Ex. 3 at 104.

Dr. Steinman referenced the March 26, 2014 EMG/NCS study as having "abnormal findings suggestive of a more proximal demyelinating lesion." The study showed prolongation of the tibial F-wave and an absent right peroneal F-wave. Dr. Steinman described F-waves as the ability of the nerves in their more proximal part to conduct electricity. Absence of F-waves means a response cannot be elicited at all; prolonged F-waves are abnormal but elicitable. Absence is indicative of peripheral nerve disorder, but of a more proximal than distal variety. Tr. 567-68. Since CIDP is an inflammatory demyelinating polyneuropathy with prolonged or absent F-waves, the impression was "inflammatory neuromuscular disease likely." Tr. 197-98; Pet. Ex. 89 at 36. Dr. Steinman conceded the findings were unsupportive of CIDP or GBS, but while Dr. Hu found the EMG unremarkable, Dr. Knox said it suggested demyelination.[126] Tr. 222-23, 287; Pet. Ex. 3 at 140; Pet. Ex. 13 at 181; Pet. Ex. 55 at 3.

Dr. Steinman stated that even if Dr. Knox was questioning the diagnosis, he still referred to the diagnosis as CIDP three years later. Tr. 198. In his "personal opinion," though Dr. Knox was struggling, he "landed on CIDP" and Dr. Steinman "commended [Dr. Knox] for trying very hard and I think he finally felt CIDP was a tenable diagnosis, but that's speculation on my part." Tr. 556. According to Dr. Steinman, Dr. Knox kept "pushing and worrying" about whether to prescribe IVIG, so he sent him to Dr. Katz. Tr. 556-57.

Dr. Steinman was critical of Dr. Katz, his opinions and failure to conduct a sensory examination on August 25, 2017. Tr. 281-82, 288-89; Pet. Ex. 89 at 71. In his view, Dr. Knox accepted Dr. Katz's opinion that petitioner did not have CIDP based on a "bad examination." Tr. 283-84. However, he pointed out that even after Dr. Knox no longer thought petitioner had CIDP, his record reflects "inflammatory or pseudoneurological disease, not CIDP. Could this be an immune joint disease?" and he sent petitioner for rheumatologic follow-up. Tr. 282-83; Pet. Ex. 89 at 79. Dr. Scalapino conducted an exam and "put it to rest" that it was not rheumatologic disease.[127] Tr. 557.

Dr. Steinman referred to Dr. Latov's March 2018 examination as "exemplary," with an extensive sensory examination showing "striking" findings of generalized weakness and sensory loss of unclear cause. Dr. Latov ordered another EMG/NCS, after which he concluded "demyelinating polyneuropathy, probably CIDP, although other causes of demyelinating polyneuropathy . . . should also be considered." Tr. 199-201, 289; Pet. Ex. 58; Pet. Ex. 63 at 4. Dr. Steinman opined that Dr. Latov put CIDP at the top of his list of possible diagnoses, referring to

[126] This in incorrect. In fact, Dr. Knox noted in the record he was puzzled that there was never any demyelination seen on testing. Pet. Ex. 13 at 315.

[127] Dr. Scalapino's assessment was neuropathic pain with poor balance, "it is not clear to me whether he has CIDP, CIDP related to diabetes, or some other neuropathy." He noted that the EMG is lukewarm support, his reflexes are intact, and that it is some immunological process because he temporarily responds to IVIG and steroids. Dr. Scalapino considered a nerve biopsy. Tr. 411-14; Pet. Ex. 89 at 87-95, 341-49.

him as a "real expert" who could not stop at CIDP, so he included other esoteric possibilities. Dr. Steinman added Dr. Latov to his list of "board-certified" neurologists diagnosing petitioner with CIDP. Tr. 201, 203, 290-91, 572; Pet. Ex. 63 at 4. When asked what Dr. Latov meant by "probably" CIDP, Dr. Steinman responded, "I didn't ask him," later testifying that when doctors use "probably, possibly and all of that" it is in a different context: "I think he is saying it's CIDP in my opinion." Tr. 291, 572-73.

Dr. Steinman explained that Dr. Latov's finding of no vibration or sensory loss below the clavicle is often related to the cervical spinal cord, not peripheral nerve disorder. He stressed, however, that the EMG done at Dr. Latov's request was abnormal with some mild clonic reinnervation in the distal muscles, which could be secondary to either a chronic inflammatory demyelinating neuropathy or diabetes. Tr. 570-71.

Dr. Steinman submitted that, after examining petitioner, Dr. Latov felt Rituxan, a monoclonal antibody used for treating CIDP and approved for various other diseases and off label uses, was needed even though both IVIG and Rituxan are immunoglobulins. Dr. Steinman could not comment on if petitioner's IVIG was discontinued due to his kidney disorder. Tr. 564-65.

Dr. Steinman referred to Dr. Brown's July 2018 examination as also concluding petitioner had CIDP, stating that she wrote that the EMG showed chronic sensory motor neuropathy with demyelinating features[128] and ordered IVIG for five days. Tr. 210; Pet. Ex. 59 at 34-36.

Further, Dr. Steinman stated Dr. Xiong also had a working diagnosis of CIDP, although he wrote the underlying diagnosis was "not quite clear" and petitioner's diabetes could be partially responsible. Dr. Xiong sent petitioner for another EMG and concluded it was CIDP. Dr. Steinman remarked there were now seven board-certified neurologists who diagnosed him with CIDP. Tr. 292-93: Pet. Ex. 65 at 20.

Dr. Steinman concluded that Drs. Katz,[129] Seminer, Latov, and Brown each treating petitioner with IVIG was proof he was suffering from CIDP but added "the diabetes could have caused the CIDP, but I think it's much more likely that the vaccine did it." Tr. 203.

2. Petitioner's diagnostic test results

Dr. Steinman attributed petitioner's normal EMG/NCS results to IVIG and steroid treatments. He relied on a study on whether IVIG treatment would improve electrophysiology and motor function that concluded an overall change in the measure of nerve conduction in CIDP correlated with clinical response to treatment. Tr. 204; Pet. Ex. 86. Dr. Steinman conceded the study discussed IVIG, not steroids, but stated it was still supportive. Tr. 204-05, 551-52. According to Dr. Steinman, petitioner's first EMG on January 24, 2014 was essentially normal because the steroids he took prior to testing affected the outcome. Tr. 204-05, 551-52. He then reasoned that petitioner's EMG study was abnormal on March 26, 2014, when he was receiving

[128] While Dr. Brown's impression does state that his EMG/NCS study showed "evidence of a chronic sensorimotor neuropathy with demyelinating features", her records only reference a "prior diagnosis of CIDP", as reported by petitioner. Pet. Ex. 59 at 28, 36.

[129] Dr. Steinman was referring to Dr. Knox not Dr. Katz. *See* Pet. Ex. 85.

immunoglobulins three times monthly, because his results would have been worse had he not been on IVIG. Tr. 205-06.

Dr. Steinman relied on the EMG performed in 2018 at UC Davis, with moderate motor and sensory peripheral neuropathy with demyelinating and axonal features superimposed on carpal tunnel syndrome, as showing CIDP. Tr. 206-09. He did not acknowledge that this EMG was done more than six years after petitioner's flu vaccine.

Dr. Steinman disagreed that the EFNS/PNS Guidelines are "globally accepted" or that petitioner's failure to satisfy the EFNS criteria for CIPD was determinative that he does not have CIDP. According to Dr. Steinman, the EFNS is proposed criteria and a "work in progress" to make it easier to capture CIDP patients. Tr. 305-06; Resp. Ex. A, Tab 2. He agreed the EFNS is peer reviewed, but Stanford does not accept it and UC Davis doesn't appear to use the guidelines either. Tr. 306, 313. Dr. Steinman stated that practicing doctors do not "wait around" for all the criteria and "boxes to be ticked" when dealing with a sick patient, adding he does not use or teach the EFNS criteria; he teaches diagnoses based on information. "I've been in this court too many times, and whether we are talking about the Brighton Criteria for ADEM or this criteria it is not what we do in practice." Tr. 214, 216, 306, 313. He added none of the five doctors that diagnosed petitioner with and treated him for CIDP mentioned the EFNS criteria. Tr. 215.[130]

Dr. Steinman remarked that petitioner's treating neurologist did not test him for ganglioside antibodies and any suggestion that he undergo testing not suggested by his physicians places an undue burden on petitioner. Tr. 261; Pet. Ex. 74 at 2. In any event, it is too late to test him now because it is unknown whether the antibodies disappear or persist after causing the disease. He stated neither side filed any literature addressing the issue, but the answer is unknown to medical science. Tr. 262. Later, when it was noted to Dr. Steinman that petitioner tested positive for ganglioside antibodies on November 20, 2018 at UC Davis, Dr. Steinman reasoned that, potent immune-suppressant drugs, IVIG, steroids, and Imuran can give a false positive. Thus, those test results are not indicative of what the result would have been during the triggering period. Tr. 268-69; Pet. Ex. 65 at 7-8.

Dr. Steinman agreed that petitioner's CSF level, though mildly elevated at 73, was low for CIDP, but again stated his treatment may have affected the lab values toward normal. He agreed that elevated CSF has other potential causes, including diabetes. Tr. 552-53. He agreed a diagnosis of CIDP cannot be based on protein level alone and acknowledged that petitioner's spinal fluid did not contain many leukocytes, or the cells that help the body fight infection, but argued the CSF was still consistent with inflammatory neuropathy. He disagreed that an absolute value for protein level in CIDP should be over 100, particularly when the patient is receiving treatment. Tr. 557-58.

Dr. Steinman further submitted that petitioner's treatment affected his reflexes, which was why his reflexes were sometimes absent in many locations, sometimes normal, and sometimes only in the lower extremities. "When therapy is given to make a person better, often lab tests

[130] Dr. Steinman is incorrect. The medical record for Dr. Xiong at UC Davis reflects "[R]epeated EMG showed both demyelinating and axonal loss features, but it did not meet the EFNS electrophysiological criteria of CIDP." Pet. Ex. 107 at 9-10. Further, petitioner's IVIG was denied because he did not meet the EMG criteria pursuant to the EFNS. *See* Pet. Ex. 24.

improve and the clinical exam improves, and that's I think what we saw and is the underlying explanation." Tr. 552, 567. Dr. Steinman disagreed that those suffering from CIDP have no reflexes and that reflexes remain abnormal even after treatment, "I think you can have reflexes, especially if the treatment were somewhat effective and begun early enough in the course of the disease, that you could get them back." Tr. 568.

Dr. Steinman stated petitioner's presenting complaint in September 2013 of pain throughout the body is indicative of CIDP because the disease starts with severe pain caused by acute inflammation. Tr. 561. He conceded, however, that it is difficult to distinguish myalgic pain, joint pain, and neuropathic pain since they all have certain commonalities and specificities due to the intimate and intricate relationship between peripheral nerves and joints. Tr. 558-59. He relied on literature describing three patients with joint mobility impairment and pain in the limbs and skeleton as early features of GBS, stating this would apply to CIDP as well. Tr. 559; Pet. Ex. 96.[131]

Dr. Steinman stated that petitioner's muscle enzymes were normal. Though a muscle biopsy was not done, petitioner did not have chronic steroid-responsive muscle weakness, and further, steroid-responsive myopathy would not explain the demyelinating findings on EMG and abnormal CSF with mildly elevated proteins, which is consistent with inflammatory neuropathy. Tr. 218; Pet. Ex 74 at 2. Dr. Steinman suggested that this was Dr. Bhat's thinking in 2013 when he documented elevated CSF protein, lymphocytes, and white blood count and provided a differential diagnosis of aseptic meningitis, which could be a reaction to the vaccine. The CT of the lower extremities and the muscle enzyme aldolase were normal, making inflammatory polymyositis unlikely. Tr. 220-21.

Dr. Steinman agreed petitioner's complete recovery following IVIG treatments was atypical of CIDP but stated petitioner's case is not typical. Tr. 226; Resp. Ex. D. He then added he has seen some dramatic responses to IVIG early in the disease but admitted petitioner's visit with Dr. Brown in July 2018 was not early in the disease, though it would not preclude petitioner's response as being real. Tr. 570. Dr. Steinman disagreed that IVIG could provide a strong placebo effect. Tr. 577.

Dr. Steinman stated that a lot of diagnoses were "thrown out", but CIDP remained constant. Tr. 221. "Overall and unmistakably, five doctors come up with CIDP" and "to take all of the information and say nah, it's not CIDP, in my opinion, is wrong. I think it is CIDP." Tr. 223.

c. Dr. Chaudhry's Opinion

While he acknowledged petitioner's suffering, Dr. Chaudhry maintains that a definitive diagnosis of CIDP was never established. He noted that petitioner's treating physicians ordered muscle biopsies and MRIs of the spine to evaluate for muscle disorder and spinal cord pathology, but neither were ever done, and petitioner's treatment was stopped over a year prior to the hearing. Resp. Ex. A. Certainly, Dr. Knox gave a lot of thought to his decision to stop treatment, as no doctor puts themselves at risk by stopping treatment of a patient who needs it, especially when the patient requires two canes and a wheelchair. However, based on all the evidence filed in this matter

[131] I. Soryal et al., *Impaired joint mobility in Guillain-Barre syndrome: a primary or a secondary phenomenon?*, 55 J. NEUROL. NEUROSURG. PSYCHIATRY 1014 (1992), filed as "Pet. Ex. 96."

and his own expertise and experience in neurology, there is no support for a diagnosis of CIDP or GBS. Tr. 321-23, 351-53, 440-41. Dr. Chaudhry concluded that, without a definite diagnosis of CIDP, the relationship between the flu vaccine and CIDP cannot be addressed. He submitted that flu vaccine generally has not been associated with CIDP, though the Institute of Medicine ("IOM") identified five publications reporting CIDP after flu vaccine and concluded that there was inadequate evidence to accept or reject a causal relationship. Resp. Ex. A at 9.

Dr. Chaudhry described CIDP as a chronic disease thought to have an inflammatory basis causing the myelin sheath not to function.[132] CIDP presents with symptoms of numbness and tingling followed by a pattern of weakness in the proximal and distal muscles in symmetrical fashion over a two-month period. Findings on examination include sensory loss, weakness, and generalized loss of reflexes. Spinal fluid protein is elevated without an elevation of white blood cells, called albuminocytological dissociation. EMG testing classically shows demyelination, which is indicative of the nerve not working correctly. Currently, there are newer techniques for diagnosing CIDP, including MRIs to show thickened or enhanced nerves in various areas; ultrasound to look at the same nerves for thickening or enlargement; and muscle biopsies which show inflammation and demyelination. The diagnosis starts with clinical presentation and is confirmed by laboratory testing. CIDP is a very well-defined syndrome that is often over-diagnosed. There are various immune treatment modalities including IVIG, prednisone, and plasma exchange, which are 70 percent effective. Tr. 323-25.

Dr. Chaudhry stated a diagnosis of CIDP is not based on a patient's response to IVIG. In *Allen*,[133] a study of 60 patients diagnosed with CIDP, only 47 percent met the criteria for CIDP, and 50 percent met the EFNS criteria. Diabetic peripheral neuropathy was one of the highest alternative diagnoses, and mild elevated proteins were a factor leading to misdiagnosis of CIDP. The study revealed that many patients who were treated with IVIG and corticosteroids for years never had CIDP. Tr. 331-34; Resp. Ex. E at 5. [134]

Dr. Chaudhry referred to another study in which Neurofactor, an infusion company, and Dr. Katz were involved. The study showed only 30 percent of 250 patients studied receiving IVIG for CIDP were appropriate candidates for IVIG, with only 15 percent meeting the electrodiagnostic criteria. Dr. Chaudhry agreed IVIG is serious treatment with serious side effects, but patients think they are getting better and when it is stopped, they feel worse due to a placebo effect. Tr. 335-36; Resp. Ex. F.[135] According to Dr. Chaudhry, the study was an "eye-opener" since the infusion company, who makes money selling the treatment, was doing the research. Tr. 336-37.

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[132] Myelin is the insulation on top of the nerves. Demyelination refers to when the myelin sheath is not working. Tr. 323.

[133] Jeffrey A. Allen & Richard A. Lewis, *CIDP diagnostic pitfalls and perception of treatment benefit*, 85 NEUROLOGY 98 (2015), filed as "Resp. Ex. E."

[134] CIDP treatment in these cases was thought to be partially helpful but also blinded the treating physicians to diagnostic errors and perpetuated suboptimal treatment of the immune-mediated condition.

[135] Todd D. Levine et al., *Review process for IVIg treatment*, 8 NEUROLOGY: CLINICAL PRAC. 429 (2018), filed as "Resp. Ex. F."

1. Petitioner's EMG and other testing

Dr. Chaudhry is an expert in electrodiagnostic medicine geared toward EMGs, EEG, and other physiological procedures. He conducts and interprets between 100 and 200 EMGs each month. Tr. 319-321.[136] In anticipation of the hearing, Dr. Chaudhry created a chart of all petitioner's EMGs results. Tr. 338; Resp. Ex. D; Resp Ex. G. At the time of hearing, petitioner had undergone eight EMGs performed between January 2014 and November 2018. Tr. 339.

Dr. Chaudhry provided a complete recitation of the electrodiagnostic criteria associated with CIDP, breaking down the criteria into "Definite CIDP", "Probable CIDP", and "Possible CIDP", and concluding that none of the studies performed on petitioner met the criteria. Resp. Ex. D at 8-9. He added that petitioner also did not meet the supportive criteria of elevated CSF protein with leukocyte count of less than 10 cells/mm, as he had 29 white blood cells in the CSF. *Id.* at 9. According to Dr. Chaudhry, petitioner did not and does not have CIDP.

Dr. Chaudhry stated the EFNS provides the criteria for CIDP and is the gold standard for diagnosis. The EFNS is used by insurance companies for approval of IVIG, even if the criteria is not referred to by name. Further, just because a physician does not document the record as applying the EFNS criteria does not mean it was not used. Tr. 325-28. In this case, petitioner's IVIG was stopped due to his failure to satisfy two of the EFNS criteria, which includes reduced conduction velocity, prolonged latency, and prolonged F-waves. Tr. 525; Pet. Ex. 24. Dr. Chaudhry agreed additional IVIG was approved after petitioner appealed. Tr. 544.

Dr. Chaudhry described EMG testing as the most liberal of the EFNS criteria available and the "bread and butter" of neurology. He explained that evidence of demyelination on EMG/NCS studies is required to confirm a diagnosis of CIDP. Demyelination occurs when there is blockage of conduction; when the myelin sheath on the nerve is not functioning, the nerve will not conduct messages as fast as it should. Latency, or the time it takes for the impulse to travel from one end to the other, will be prolonged with demyelination. Conduction velocity is how fast the message travels along the course of the nerve, with distance divided by the latency to show the reduction. Tr. 326-27.

According to Dr. Chaudhry, when "demyelination" is seen on EMG, the report will state "this is consistent with CIDP or . . . this is GBS" or a list of other diseases such as MAG, POEMS, or diabetic neuropathy. Tr. 327, 339-340. When an EMG is at the lower limits of normal, the clinical picture must be assessed, including spinal fluid results and the clinical course thus far. Every laboratory has a different lower level of normal value, but the criteria applied is the EFNS for both clinical practice and research. Tr. 329-331.

Dr. Chaudhry was surprised that an EMG was not done until January 17, 2014, four months after the presentation of symptoms and three months into treatment. Tr. 339-340. NCS is a stimulation test of both motor and sensory nerve conduction. Motor conduction tests the median, ulnar, peroneal, and tibial nerves, and petitioner's motor conduction tests show most of his doctors

[136] Dr. Chaudhry is a neurologist and board certified by the American Board of Psychiatry and Neurology in neurology, clinical neurophysiology, and neuromuscular medicine, and by the American Board of Electrodiagnostic Medicine. All pertain to the diagnosis, treatment, evolution, and electrophysiology of CIDP and neuropathy. Tr. 320-21.

tested only the right side, though some also tested the left side. Sensory nerve conduction tests the median, ulnar, superficial peroneal, sural, and radial nerves. The January 17, 2014 EMG/NCS study was normal for all sensory amplitudes of the nerves. The motor conduction velocity showed some slowing in the median nerve reflected as right-sided carpal tunnel syndrome. Tr. 342. The motor nerve conduction, latencies, and amplitudes were normal, with no evidence of demyelination in either the axon or the myelin. Tr. 342.[137]

Dr. Chaudhry described F-waves as the nerve messages traveling to and from the spine. The January 17, 2014 EMG/NCS showed one F-wave abnormality measuring 65.6.[138] Tr. 342; Pet. Ex. 13 at 195. This EMG was consistent with diabetes and/or a bad back, but not demyelinating neuropathy. Tr. 342-43. He referred to Dr. Seminer's conclusion that this EMG was unsupportive of either GBS or CIDP and evidence of any neuropathy was unclear noting that Dr. Knox reached the same conclusion multiple times after repeat EMG/NCS studies. Tr. 343.

Dr. Chaudhry referenced the March 26, 2014 EMG as showing prolongation and absence of lower limb F-waves with more proximate demyelination, which was "very mildly abnormal," but common for someone with back issues. Further, it did not meet EFNS criteria for a diagnosis of CIDP. Tr. 344; Pet. Ex. 13 at 181. The November 18, 2014 EMG/NCS testing was noted as normal and unchanged from the March study; the prolongation of the lower limb F-wave was noted again but still was not within the EFNS range for CIDP. Tr. 344; Pet. Ex. 13 at 341.

Dr. Chaudhry stated the June 24, 2015 study did not include the data but based on the narrative report the right upper and lower limbs, including the F-waves, were acceptable when compared to the testing in March 2014. Tr. 344-45; Pet. Ex. 20 at 47; Pet. Ex. 20 at 101. He stated that the needle portion of an EMG is excellent in distinguishing between a nerve disorder and a muscle disorder because muscle activity recruitment is much quicker than nerve disease. When there is a weak muscle due to damaged or blocked nerves, the needle picks up on it regardless of which muscle is tested. The June 24, 2015 EMG was normal for motor and recruitment, which is not consistent with CIDP or any neuropathy. Tr. 345. This EMG suggested a small motor unit of the right leg, which is generally indicative of muscle disease, disuse, or statin usage, which can cause weak muscles. Petitioner's CPK was normal, so polymyositis was ruled out, but concern for sarcoid was noted. A muscle biopsy should have been done. Tr. 348-49.

Dr. Chaudhry stated that during both the March 24, 2014 and June 24, 2015 EMG/NCS testing, Dr. Knox also performed a repetitive nerve stimulation study (RNS) and antibody testing, which generally is not done unless there is concern for myasthenia gravis. Had Dr. Chaudhry done the testing, he would have done a single fiber EMG, which is very sensitive for myasthenia gravis, because there was mention of head drop on examination and head drop is a sign of myasthenia gravis. The EMGs were indicative of a muscle condition, but not CIDP. Tr. 349-351.[139]

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[137] Latency refers to how long the nerves take to travel, and amplitude refers to how big the response is. Tr. 342.

[138] The EFNS criteria for a diagnosis of CIDP is 72. Tr. 342.

[139] Dr. Steinman adamantly disagreed with Dr. Chaudhry that petitioner's symptoms were more consistent with myasthenia gravis ("MG") despite Dr. Steinman having extensive knowledge of the disease and publishing on it. MG is a disease against the acetylcholine receptor at the neuromuscular junction. Those with the disease have many of the symptoms that petitioner described. Dr. Steinman testified in *D.G.* that MG has a few other antigenic targets, e.g., muscle-specific kinase, but he does not know that any of these other antigenic targets is a direct molecular mimic of anything in influenza vaccine that cross-reacts with myelin. He stated myasthenia gravis is probably one of the best

Dr. Chaudhry stated the December 20, 2017 EMG showed worsening left-sided carpal tunnel in need of surgery. Tr. 346; Pet. Ex. 89 at 180.

Dr. Chaudhry referenced the EMG studies in June and November 2018 as showing "demyelinating features." Dr. Langsdorf performed the June 2018 study and wrote "abnormal study. There is electrophysiological evidence for chronic sensorimotor polyneuropathy with demyelinating features." Dr. Chaudhry stated the use of the word "features" suggests "nerve conduction velocity being reduced." Dr. Maselli performed the November 2018 study performed by Dr. Maselli showed moderate sensory neuropathy with both demyelinating and axonal "features," but not CIDP, which requires more severe demyelination, a much higher reduction in velocity, a much greater change in F-wave latency and distal latency, and much more conduction block. Tr. 347-48.

Dr. Chaudhry concluded that none of the eight EMGs petitioner underwent over a four-year period met the EFNS criteria to support a CIDP diagnosis, though the last two had features "consistent with someone who's had diabetes for so many years." Tr. 348; Resp. Ex. G.

In summary, Dr. Chaudhry stated the January 2014 EMG did not support GBS or CIDP. Though the March 2014 EMG showed proximal demyelination because of the F-waves, this was related to petitioner's chronic back issues, not CIDP. The November 2014 EMG was unchanged from March. The June 2016 EMG suggested myopathy but was inconsistent for CIDP or neuropathy. The August 2016 and December 2017 EMGs showed no change from June 2016. Tr. 533-34. However, the June 2018 EMG showed mild changes to sera responses which had previously been normal, and Dr. Langsdorf concluded there were mixed features of sensory disorder neuropathy with demyelinating features, which is a common finding in patients with diabetes with or without neuropathy. Tr. 534. Dr. Chaudhry added the data from the June 2018 EMG was hard to reconcile because the next EMG in November 2018 was improved. None of the studies met the criteria for CIDP but were typical for patients with diabetic neuropathy-related symptoms. Tr. 535.

Dr. Chaudhry disagreed that petitioner's treatment caused his EMG/NCS results to appear normal, noting that the test results were normal even when he was not being treated. Further, the article relied on by Dr. Steinman showing improvement of EMG results with IVIG treatment also concluded that EMG results for patients with GBS are never normal, even years after they have recovered. Tr. 353-54; Pet. Ex. 86. He concluded when an EMG shows a mixture of axonal loss and mild demyelination on EMG, "invariably the diagnosis is diabetes." Tr. 355.

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understood of all autoimmune conditions. Some interesting molecular mimics between other viruses, but not influenza virus, exist such as herpes virus and acetylcholine receptor as a molecular mimic. *See D.G.*, *supra* note 83, at *129. Yet here, he refused to discuss MG, even though Dr. Chaudhry opined it could explain petitioner's symptoms including head drop, muscle weakness, recurrent bouts after IVIG treatments, and ongoing medical issues in combination with his co morbidities. Dr. Steinman avoided the topic criticizing both Dr. Chaudhry and the undersigned for questioning why petitioner had not undergone the testing suggested by his treaters that could determine his ongoing condition and assist in his treatment. Based on Dr. Steinman's admission in other cases that there are no known antigenic targets in MG that are a direct molecular mimic in flu vaccine that cross reacts with myelin, acknowledging that some of petitioner's symptoms fit with a diagnosis of MG would not have bode well for petitioner in this case.

2. Petitioner's medical history

In furtherance of his opinion that petitioner does not have CIDP, Dr. Chaudhry discussed petitioner's medical records at length and highlighted various findings and references in the medical records that are inconsistent with a diagnosis of CIDP.

Dr. Chaudhry noted that one month after the flu vaccine, at petitioner's initial visit with Dr. Hopkins on September 17, 2013, the neurological examination was non-focal except for pain and slow movements. Dr. Hopkins's assessment was acute arthritis, and he prescribed a steroid. Both CIDP and GBS present with numbness, tingling, and a pattern of weakness that starts at the feet and ascends, but petitioner had none of these complaints at this visit. Tr. 366-67.

Dr. Chaudhry stated petitioner then presented to the ER reporting debilitating joint pain that started a day after the flu vaccine which was relieved by prednisone. GBS does not respond to prednisone and, if anything, prednisone worsens GBS. Petitioner complained of severe pain and weakness throughout his body, but the weakness was not ascending as it is with GBS and CIDP. Further, while pain can be a feature of neuropathies, it is not a presenting symptom of CIDP. Dr. Mahmood wrote that steroids made a "night and day difference," which would not occur in GBS or CIDP. Petitioner had grip weakness that was partially due to pain and some weakness in the proximal muscles, but his sensory examination was normal. He had a hint of ptosis and head drop, which are presenting signs of myasthenia gravis. Dr. Mahmood wrote no ataxia despite generalized weakness and recommended IVIG and continuation of steroids even though GBS is not treated with steroids. Petitioner had unexplained elevated CPK/CRP at 43.9, which do not elevate with GBS or CIDP. Tr. 367-370. Dr. Mahmood assessed petitioner's symptoms as suggesting the possibility of a post-vaccination type of GBS but noted that GBS would not explain his muscle tenderness and soreness. Pet. Ex. 3 at 27.

Dr. Chaudhry stated petitioner was hospitalized again four weeks later, at which time he denied numbness. Pet. Ex. 3 at 60. There was some weakness of the upper and lower extremities of 4+/5, but petitioner's greater weakness in the shoulders and thighs indicated the proximal muscles were more affected than the distal muscles. The proximal weakness of the hip and shoulder girdle were indicative of myasthenia gravis, and an EMG/NCS study was not ordered because the doctors were not concentrating on neuropathy. Dr. Chaudhry referenced the fourth page of the medical record which noted polymyositis, serum sickness, neuromuscular disease vs. myopathy, aseptic meningitis, and steroid responsive myopathy of unclear etiology. There was no mention of GBS or CIDP in the record. Tr. 370-74.

Dr. Chaudhry referred to the petitioner's November 12, 2013 medical visit, which documented that a muscle biopsy was considered but not done. Tr. 374-75.

Dr. Chaudhry stated petitioner complained of pain in all joints at his December 9, 2013 visit with Dr. Seminer, but joint pain is not associated with GBS or CIDP. Petitioner's reflexes were intact except for his ankles with distal loss to pin and vibration, but all reflexes should be reduced or absent in GBS and CIDP. An EMG was ordered for the first time. Tr. 374-75. Dr. Seminer's assessment was "probably CIDP." Tr. 504.

Dr. Chaudhry stated petitioner again came under the care of Dr. Mahmood when he was hospitalized on January 8, 2014. His reflexes were still normal. Tr. 518; Pet. Ex. 100 at 2. Dr. Mahmood wrote that his course was now chronic and should be considered CIDP. Tr. 501; Pet. Ex. 100 at 12. Later, Dr. Chaudhry read Dr. Mahmood's complete entry in the record, which stated:

> …it's become chronic and should be considered CIDP, however, the muscle stiffness, tenderness and soreness that the patient describes are more suggestive of myositis or myopathy. His steroid responsiveness remains quite impressive. This clouds the diagnosis. I am still concerned about the possibility of spinal cord involvement. He has not been able to get the MRI because of the device...the patient's diagnosis remain[s] elusive.

Tr. 519-520; Pet. Ex. 100 at 12-13. Dr. Chaudhry also pointed out that the discharge summary reads, "patient coming in with weakness secondary to steroid, responsive proximal myopathy of one clear (sic) etiology." Tr. 518; Pet. Ex. 100 at 8.

Dr. Chaudhry described the January 17, 2014 EMG as non-diagnostic. Dr. Seminer suggested a muscle biopsy and MRI for myasthenia gravis and muscle disease. Tr. 376. On February 24, 2014, petitioner had slight weakness in his grip and legs and deep reflexes were absent only in the lower extremities, and IVIG was started for "probable CIDP". Tr. 376-77.

Dr. Chaudhry stated that when petitioner saw Dr. Hu on March 24, 2014 his strength was normal, with decreased pinprick only on the left arm and leg. Dr. Hu wrote "potential CIDP" but the EMG is unremarkable and recommended repeat testing for myasthenia gravis, repeat antibody testing, and an MRI to rule out cervical cord myopathy. Dr. Chaudhry stated that repeat CSF protein elevation at 73 meant nothing in the setting of diabetes and the EMG pointed to myasthenia gravis. Tr. 377-78, 505-06. He agreed that Dr. Hu's assessment was "potential CIDP," but stated the whole examination must be considered, not just "selective issues." Dr. Hu was questioning the diagnosis and petitioner's other physicians "were clearly not convinced that this is entirely CIDP." Tr. 505-06.

Dr. Chaudhry stated petitioner presented at his next visit with Dr. Seminer with complaints of fatigue, difficulty swallowing and speaking, and had ptosis and head drop noted again, which are not symptoms of CIDP. Dr. Seminer recommended a small fiber EMG. Tr. 380.

Dr. Chaudhry referred to Dr. Knox, who took over petitioner's neurological care on May 9, 2014, as being in the best position for diagnostic assessment since he saw him over a four-year period until August 10, 2018. Pet. Ex. 5 at 4-12; Pet. Ex. 99. At his first visit, Dr. Knox noted trace ankle and knee reflexes, with five minus weakness of right toe curling. Brachioradialis and triceps were one plus. There was slight difficulty with pinprick and vibration was seven seconds. Dr. Chaudhry explained demyelinating conditions like GBS or CIDP affect vibration sense, but the large fibers showed no demyelination, so this is not CIDP. Tr. 380-82; Pet. Ex. 13 at 243-45.

Dr. Chaudhry noted at petitioner's examination on July 17, 2014 he was improved on the left; he could lift his arms higher, and had no issues getting up, but had a slight foot drop with no clear weakness. Tr. 383-84; Pet. Ex. 13 at 272.

Dr. Chaudhry stated petitioner was noted to have "giveaway" weakness on October 11, 2014, which could be due to pain or issues with balance, though it is often voluntary. Tr. 385-86; Pet. Ex. 13 at 301.

Dr. Chaudhry remarked that in Dr. Knox's note for November 18, 2014, he wrote that it was interesting that petitioner's nerve conduction study "never showed demyelination???" According to Dr. Chaudhry, there should have been "marked findings of demyelination" after a year. Tr. 387-88; Pet. Ex. 13 at 311.

Dr. Chaudhry stated Dr. Knox ordered another EMG on July 24, 2015, and again noted concern for primary muscle disorder and the need for muscle biopsy and a second opinion because he was now questioning himself. This, Dr. Chaudhry added, is not a "clear case of CIDP" as suggested by Dr. Steinman; his own treater was questioning what is going on. Tr. 388-390; Pet. Ex. 20 at 46-47. Dr. Chaudhry noted that "giveaway weakness" without bulk loss was documented again on January 26, 2016. Tr. 390-92; Pet. 20 at 94.

Dr. Chaudhry referred to petitioner's March 18, 2016 visit at which muscle disease was again considered and it was documented that it was unclear whether petitioner had sensory deficit or weakness, since he appeared to confuse the two. Tr. 392-93; Pet. Ex. 20 at 103. The thought at that time may have been myopathy due to his urinary incontinence, but myopathy does not have numbness. It was again noted that an MRI could not be done due to a stimulator in his back. He also had cervical spondylosis. The examination was "normal". Tr. 393-95; Pet. Ex. 2 at 109.

Dr. Chaudhry referenced petitioner's visit on August 18, 2016, where Dr. Knox wrote prolonged F-wave and edge reflexes from diabetes, or from the spine, but "not CIDP". Tr. 395-96; Pet. Ex. 20 at 148. Insurance denied IVIG because he did not meet the EFNS criteria. Tr. 396-97.

Dr. Chaudhry stated petitioner's complaints of leg and head heaviness and his arms feeling asleep at his visit with Dr. Knox on July 19, 2017 are common myasthenia gravis complaints. Tr. 398; Pet. Ex. 89 at 43. On examination, petitioner only had weakness in the deltoid but presented in a wheelchair without medical explanation. Tr. 398-400. Dr. Knox documented two years and two months of Imuran that was discontinued because it did not work and sent petitioner to Dr. Katz due to concerns with the diagnosis and for a second opinion on treating with Rituxan. Tr. 400-01.

Dr. Chaudhry referred to Dr. Katz as highly respected and well-known to him, stating that Dr. Katz did not believe petitioner had CIDP. Tr. 401-02; Pet. Ex. 85 at 4. Dr. Katz was aware of petitioner's history and clinical presentation and attributed the leg symptoms to sciatica. He found petitioner to be tremulous with normal bulk and tone. Despite noting "giveaway", Dr. Katz was able to determine petitioner's strength as 5/5 in his both his arms and legs. Dr. Katz had the EMG/NCS results showing the sensory nerves and reflexes as normal. Dr. Katz saw no evidence of CIDP, and Dr. Chaudhry agrees. Petitioner had multiple normal EMGs by this time, his CSF protein numbers were explainable, he had reflexes and displayed "giveaway" weakness, and there was no progression, so Dr. Katz questioned why petitioner could not walk. He suggested another NCS and repetitive stimulation study for myasthenia gravis with low suspicion anything would be found. Tr. 403-08.

Dr. Chaudhry noted that Dr. Knox again documented "giveaway weakness" on August 25, 2017, no longer believed petitioner had CIDP, and discontinued IVIG treatment. Dr. Knox questioned whether he had "another inflammatory or pseudoneurological disease, something that mimics a neurological disease." Tr. 408-410; Pet. Ex. 89 at 302, 311.

Dr. Chaudhry referred to the rheumatology visit with Dr. Scalapino, during which petitioner could not lift his shoulders while sitting but had elevation to 120 or 130 degrees while laying down. Dr. Chaudhry noted several things from this record: petitioner was able to squat and rise which are difficult maneuvers with proximal muscle weakness, his reflexes were intact throughout, and he had decreased vibration in his feet. Dr. Scalapino's assessment was neuropathic pain with poor balance, but it was unclear if it was CIDP, CIDP related to diabetes, or some other neuropathy. The EMG showed "lukewarm support" for a CIDP diagnosis and reflexes were intact. Dr. Scalapino noted there was some immunological process because he temporarily responds to IVIG and steroids and considered a nerve biopsy. Tr. 412-14; Pet. Ex. 89 at 87-95, 341-49.

Dr. Chaudhry referred to Dr. Knox's examination on December 20, 2017, again documenting his concern that petitioner did not have CIDP and the need for an MRI. Tr. 418-19; Pet. Ex. 89 at 107, 115.

Dr. Chaudhry then turned to Dr. Latov's March 2018 examination, pointing out that Dr. Latov was the only doctor to record sensory loss, which Dr. Chaudhry noted is subjective because patients are asked and answer questions on sensory examination. After this examination, Dr. Latov wrote generalized weakness and sensory loss. Tr. 419-420, 423. Then, after the EMG he ordered, Dr. Latov wrote demyelinating polyneuropathy, probably CIDP, but also added other potential causes of demyelinating neuropathy including MAG, POEMS and CMT-1. Tr. 513-14.

Dr. Chaudhry noted that petitioner presented to the ER and came under the care of Dr. Brown, who also documented "giveaway" on examination but assessed him with CIDP and ordered IVIG for five days. After treatment, petitioner reported complete resolution of symptoms, which Dr. Chaudhry stated does not happen with CIDP. Tr. 420-21.

Dr. Chaudhry noted petitioner's return to Dr. Knox on August 10, 2018, at which time Dr. Knox noted giveaway weakness "in all muscles," and ratcheting effect, which is like giveaway weakness but more consistent with superimposed functional pain. Tr. 522; Pet. Ex. 99 at 5. Dr. Knox continued to question the diagnosis, wondering if it was coming from petitioner's neck since the EMG showed carpal tunnel or if it was immune joint disease. Dr. Knox wanted the stimulator removed so an MRI could be done, and he discontinued IVIG again. Tr. 531-32; Pet. Ex. 99 at 10.

Dr. Chaudhry stated Dr. Xiong also documented the underlying etiologies as "not quite clear" in the fall of 2018. Dr. Xiong's assessment included that diabetes could partially contribute to the neuropathy, but it was unusual for diabetes and neuropathy to fluctuate so much. He ordered a repeat EMG, GM1 antibody testing, and considered nerve and muscle biopsy. Tr. 423-24.

d. The Record Supports a Diagnosis of CIDP

Petitioner has a lengthy history of comorbidities, including but not limited to uncontrolled Type 2 diabetes with peripheral neuropathy and vascular compromise; five spinal surgeries with implantation of a now dysfunctional spinal cord stimulator that is embedded in tissue and suspected of causing cord compression; cervical spine issues with radicular pain and carpal tunnel syndrome in need of surgery; ischemic attack; memory issues; syncope; urinary issues; numbness in his extremities; and foot pain prior to his flu vaccine. Following his vaccination, he suffered muscle and joint pain, stiffness, paresthesia, and weakness that responded immediately to steroid treatment. He had numerous symptom relapses following tapering or discontinuance of steroids. His examinations ranged from normal to proximal weakness, distal sensory loss, and reduced reflexes. He has not had an MRI of the lumbar spine due to the now embedded stimulator in his back or a muscle or nerve biopsy. He has had multiple EMG/NCS testing. The first four on January 24, 2014, March 26, 2014, November 18, 2014, and June 24, 2015, showed carpal tunnel syndrome and minimal abnormalities which could be associated with diabetic neuropathy. The June and October 2018 EMG/NCS studies, five years post-vaccination, showed demyelination. CSF studies showed elevated protein with cells[140] and all other testing was negative.

Petitioner has been treated with various forms of oral and IV steroids including IVIG, Rituxan, Cellcept, Imuran, and methotrexate. He has experienced complete resolution of symptoms after IVIG treatment and relapsing symptoms associated with the withdrawal of steroids. In 2017, four years after his flu vaccine, he became so debilitated he required two canes or a wheelchair for mobility. Pet. Ex. 89 at 46.

As of late 2021 when updated medical records were filed, petitioner had presented to Dr. Shaoulian at the Neurology Muscular Dystrophy and Neuropathy Institute and reported an eight-year history of CIDP initially diagnosed as GBS and treated with steroids that began 18 days after a flu vaccine. He was diagnosed with CIDP after a recurrence 47 days later. He presented with poor balance, a history of falls, and use of a cane and walker or a wheelchair for short and long distances, respectively. Pet. Ex. 111 at 6. The assessment after examination was weakness in all extremities with loss of vibration sense in the distal lower and upper extremities and decreased pin sensation in the distal lower extremities. The 2018 EMG/NCS from Cornell was read as generalized sensorimotor polyneuropathy with demyelinating features. Labs from Sutter in 2017 and 2018 were negative for monoclonal bands and he had multiple levels of foraminal narrowing. The diagnosis on that date was hyperesthesia; difficulty walking, not elsewhere classified; and weakness. *Id*. at 7.

On November 8, 2021, petitioner returned to Dr. Shaoulian for EMG/NCS testing using a bipolar needle. "Different muscles were tested to reflect different root and nerve distributions. The EMG was normal." Pet. Ex. 111 at 1. The conclusion documented "significant delayed F-waves and decreased CMAP velocities. The study is consistent with demyelinating sensory motor neuropathy." *Id*. Dr. Shaoulian also conducted an examination on that date, noting weakness in all extremities, with loss of vibration sense in the distal lower and upper extremities and decreased

[140] GBS typically has elevated protein and no cells. Pieter A. van Doorn et al., *Clinical features, pathogenesis, and treatment of Guillain-Barre syndrome*, 7 LANCET NEUROL. 939 (2008). CSF studies were performed after IVIG treatment, which is why aseptic meningitis was part of the differential diagnosis.

pin sensation in the distal lower extremities. *Id*. at 5. Dr. Shaoulian wrote that the EMG/NCS "is consistent with a demyelinating sensory motor neuropathy. Based on the patient's history, exam and workup he has CIDP." He ordered IVIG for two months. *Id*.

In weighing evidence, special masters are expected to consider the views of treating doctors. *Capizzano v. Sec'y of Health & Human Servs.*, 440 F.3d 1317, 1326 (Fed. Cir. 2006). Treating doctors' views about the appropriate diagnosis are often persuasive because the doctors have direct experience with the patient they are diagnosing. *See McCulloch v. Sec'y of Health & Human Servs.*, No. 09-293V, 2015 WL 3640610, at *20 (Fed. Cl. Spec. Mstr. May 22, 2015).

The injury ultimately alleged in this case is CIDP. Petitioner's doctors have been conflicted by his presentation throughout his treatment as shown by the records, which reflect multiple diagnoses entertained over the years, including acute arthritis, myalgia, arthralgia, polymyositis, steroid-responsive myopathy, GBS, CIDP, myelopathy with spinal cord pathology, MG, diabetic neuropathy, and primary muscle disorder. Several of these diagnoses have been ruled out, and none have been definitively confirmed. "Probable CIDP" is the only constant, and petitioner is being treated for "probable CIDP." However, treatment is not a determinative factor. Petitioner's treaters and the experts in this case agree that the medications prescribed to petitioner including IVIG, steroids, methotrexate, Imuran, and Cellcept are all used in the treatment of immune-mediated diseases generally, not only CIDP. Because petitioner has never undergone the muscle and/or nerve biopsies or MRIs recommended, no other definitive diagnosis has been made. Thus, what remains is "probable CIDP" with onset after a flu vaccination on August 19, 2013, in the context of Type 2 uncontrolled diabetes with diabetic neuropathy and vascular complication, lower lumbar injuries with possible cord compression, cervical conditions with radiculopathy, and a host of other health issues.

Therefore, having no alternative diagnosis, for the purposes of this Ruling, petitioner's diagnosis following the August 19, 2013 flu vaccine and subsequent treatment continues to be for an immune-mediated disease, "probable CIDP", with his comorbidities contributing to his disabilities.

e. Analysis of Prong II

As discussed above, petitioner has a significant history of comorbidities, the symptoms of which overlapped with those of his "probable CIDP" and clouded his presentation throughout treatment. There is preponderant evidence that petitioner's uncontrolled Type 2 diabetes with neuropathy and vascular complication caused significant pre- and post-vaccination neurological symptoms. Upon presentation to Dr. Hopkins prior to the flu vaccine in April 2013, petitioner's diabetes was untreated; he had not been under the care of a physician for well over a year and was noted to limp due to the neuropathy. Pet. Ex. 9 at 6, 10. Petitioner's treaters have also repeatedly suspected neuromuscular disease, for which petitioner has not been tested.[141] The stimulator

[141] Petitioner declared that he lost his insurance, so the ordered muscle biopsy was cancelled. He was later insured, but the testing has not been done. The records and petitioner's declaration are in conflict. The record is replete with documentation by his various treaters of the need for muscle and/or nerve biopsy, as well as MRI of the lumbar spine. Petitioner's suggestion that this is merely an electronic record repeating itself is not accurate. Further, the records indicate that the biopsies and MRI recommended were discussed with petitioner on numerous occasions, thus

implanted in his lower back has been suspected of causing cord compression contributing to his symptoms as well. Pet. Ex. 100 at 12-13. Petitioner's medical history is fundamentally at odds with a logical sequence of cause and effect that could be proposed to demonstrate that the vaccine alone was the sole cause of his "probable CIDP." Moreover, the evidence of record from petitioner's treating physicians favors the conclusion that his "probable CIDP" was not solely related to his vaccination.

As more specifically set forth above in Prong I, Petitioner has established and the Court has accepted that influenza vaccine can cause GBS and CIDP. In both GBS and CIDP, there is a known period of symptom progression which, in the case of CIDP, occurs for as many as eight weeks or more following onset.[142] It is generally understood that GBS and CIDP reach clinical nadir after about four weeks and more than eight weeks, respectively.[143] In this case, petitioner received his flu vaccination on August 19, 2013 and presented to his PCP four weeks later, on September 17, 2013, with complaints of joint pain. Neurological examination was non-focal except for pain and slow movement; he did not complain of numbness, tingling, or ascending weakness in his feet, and was prescribed a steroid. Pet. Ex. 9 at 11-13; Pet. Ex. 2 at 1-3. His first complaint of lower extremity weakness was on September 29, 2013, when he had proximal muscle weakness but normal sensory and reflexes except for his lower limbs, which is typically seen with diabetic neuropathy. Petitioner reported complete resolution of his symptoms following his September 29, 2013 hospitalization, with no additional treatment needed about five weeks after onset.

Petitioner was readmitted to the hospital on October 29, 2013 with a recurrence of symptoms. He denied numbness and his shoulders and thighs were weaker than his upper and lower extremities. Dr. Mahmood, who treated him during his prior hospitalization, now considered his symptoms consistent with a chronic relapsing and remitting course of CIDP. Pet. Ex. 3 at 60. Petitioner's course thereafter was ongoing, waxing and waning based on receipt of IVIG until 2017, when he suddenly appeared to become totally disabled and reliant on canes and a wheelchair, though his physical examinations caused his physicians to question why this was the case.

Petitioner's various treaters assessed him with "probable CIDP," diabetes with neuropathy, and suspected spinal cord compression in the lower lumbar spine, all contributing to his symptoms and debility. This is supported by objective testing over the years, which included only mildly elevated CSF consistent with diabetes, eight EMG/NCS tests (nine including the most recent in November 2021) which were essentially normal, with the first reference to demyelinating "features" five years later and in the context of diabetes and symptoms in his lower extremities consistent with cord compression, all resulting in disability.

The experts in this case are equally impressive. Dr. Steinman entered the hearing adamant both in written reports and at the start of his testimony that petitioner unequivocally had CIPD, the flu vaccine was the sole cause of petitioner's CIDP, and petitioner's diabetes and comorbidities

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discrediting petitioner's declaration that he would have undergone these tests if his doctors felt they were necessary. Pet. Ex. 110; Pet. Ex. 2 at 4; Pet. Ex. 3 at 105; Pet. Ex. 18 at 7; Pet Ex. 20 at 47, 102.

[142] Haruki Koike et al., *Paranodal dissection in chronic inflammatory demyelinating polyneuropathy with anti-neurofascin-155 and anti-contactin-1 antibodies*, 88 J. NEUROL. NEUROSURG. PSYCHIATRY 465, Fig.2, filed as "Resp. Ex. A, Tab 6."

[143] Vallat et al., *supra* note 110, at 4, 9.

played no role in his condition. However, as the hearing proceeded, Dr. Steinman's opinion evolved to include petitioner's Type 2 diabetes with diabetic neuropathy and vascular compromise as contributing to his condition. He testified that diabetes causes injury to the blood vessels that supply the nerves, and biopsies during the active phase of CIDP show inflammatory infiltrate hitting the myelin and the nerves. Both produce demyelination and sensory motor findings. In that context, petitioner already had damaged peripheral nerves from diabetic neuropathy which took a "second hit" from an inflammatory response caused by the flu vaccine via molecular mimicry. Tr. 229-230, 308, 549-550. He added that petitioner's EMGs showed an overlap of findings for diabetes and CIDP. Tr. 549-550. He concluded that "without the vaccine he would not have had this catastrophic downhill course. So it's synergized, but in this case, the vaccine was the significant aggravation because it behaves so differently than how he was going along with his diabetic neuropathy." Tr. 231-32.

Dr. Chaudhry was adamant that petitioner's clinical picture did not support CIDP. None of the recommended testing—muscle biopsy, nerve biopsy, or lower lumbar MRI—had been done, the EMGs do not support CIDP, and if petitioner's treating physicians thought he had CIDP, they would not withhold IVIG treatment. However, in addressing petitioner's Type 2 diabetes, he relied on an article which discusses a correlation between diabetes and CIDP, submitting that petitioner suffered from Type 2 diabetes with diabetic neuropathy prior to his vaccine, his glucose control was suboptimal, and his diabetic neuropathy probably progressed and worsened. Tr. 355-360. Further, Dr. Chaudhry explained EMGs of individuals with diabetic neuropathy show a mixture of slowing of nerve conduction velocity due to demyelination and loss of large myelinated fibers, as well as a decrease in nerve action potentials due to loss of axons and increased temporal dispersion. Resp. Ex. D at 7, ref. 3.[144] This was seen on petitioner's later EMGs, which showed demyelinating and axonal features consistent with diabetes and renal insufficiency, from which petitioner suffered. Resp. Ex. D at 7; Pet. Ex. 65 at 21-22. He added that part of petitioner's functional limitations may be associated with his hip arthritis and replacement. Resp. Ex. D at 7. Though rebuking that petitioner had CIDP and/or CIDP from the flu vaccine, Dr. Chaudhry seemed to suggest that petitioner may have developed CIDP caused by his diabetes. Dr. Chaudhry also agreed that IVIG helped, which meant there may be an immune-mediated component to his condition. Tr. 541-42.

f. Shyface Analysis

I must analyze this case in terms of *Shyface v. Sec'y of Health & Human Services*, in which Cheyenne Shyface was vaccinated with whole-cell DPT at the time he was beginning an *E. coli* infection. Both the DPT and the *E. coli* infection could and did cause fever, which rose to 110 degrees, resulting in his death four days later. *Shyface v. Sec'y of Health & Human Services*, 165 F.3d. 1344. Respondent defended the case and argued that the *E. coli* infection was the cause of the baby's fever and death. Cheyenne's treating physician testified that both the vaccine and the infection were equally responsible for his fever and death. The Federal Circuit held that both the vaccine and the infection were a substantial factor in causing the baby's very high fever and death and, but for the vaccination, the baby would not have had the high fever and would not have died. The Federal Circuit ruled in favor of petitioners even though petitioners did not prove that the DPT vaccine was the only or predominant cause of death. *Id*. at 1353.

[144] Said, *supra* note 122.

Similarly, petitioner suffered from uncontrolled Type 2 diabetes with neuropathy and vascular compromise, had cervical spine issues with radiculopathy and carpal tunnel syndrome, and is status post five lumbar spine surgeries with stimulator implantation and suspected cord compression prior to his flu vaccine. As Dr. Steinman ultimately conceded, the flu vaccine, in combination with already inflamed nerves from his diabetes, could have caused an aberrant response or acted in a synergistic manner, triggering an immune-mediated response and CIDP.

Dr. Chaudhry agreed that petitioner's symptoms were atypical, that his doctors were unsure of his diagnosis and considered a variety of possibilities, but petitioner continues to be treated for "probable CIPD", in combination with symptoms associated with his comorbidities.

Based on the foregoing, I find that petitioner's flu vaccination was a substantial factor in his developing "probable CIDP" and, in combination with his comorbidities, was a substantial factor in causing his current disability.

Accordingly, petitioner has sustained his burden under Prong II.

3. Petitioner Has Shown an Appropriate Temporal Relationship Between His Receipt of an Influenza Vaccine and his Development of "Probable CIPD" Only.

Dr. Steinman acknowledged the discrepancies in the record regarding onset as within one day or three weeks after vaccination. "Well, I think the onset occurred about three weeks after the shot. . . . But I mean, I'm just saying that, even force me to accept the day after, and I'll still say it's okay timewise for invoking the flu shot as a cause." Tr. 185, 187, 246. He referred to *Shonberger* as showing a 0–1-day period between vaccination and onset, stating that an onset within 24 hours is mechanistically possible with a recall response, which is when the immune system is familiar with something and responds, "really fast due to immunologic memory." Tr. 188-89; 577-79; Pet. Ex. 35 at 8.[145] Dr. Steinman, however, noted a preference for onset three weeks after vaccination, relying on Dr. Latov's history taken in March 2018 and stating it was clearer than earlier records even though it was five years after the vaccine. Tr. 186; Pet. Ex. 58. He then stated the record supported demyelinating disease within one month of immunization. Tr. 245.

Dr. Steinman was presented with Dr. Tandon's September 29, 2013 record which documented petitioner's report of a flu shot with generalized aches and pains the next day, symptoms that progressed and worsened over the next several weeks, along with his visit to Dr. Hopkins on September 17, 2013. Dr. Steinman responded, "…I think this is an exquisite description" of Mr. Davis's testimony. He stated that the generalized aches and pains petitioner felt the next day were reflective of how some people feel after the flu vaccine, and the progressing symptoms over the following weeks, "percolation, progression, latency…" that worsened until he had difficulty getting out of bed all fit with his theory of onset after one day or within a few weeks. Tr. 256-57. Dr. Steinman agreed that petitioner's symptoms occurred prior to his visit with Dr. Hopkins on September 17, 2013. Tr. 257.

[145] Lawrence B. Schonberger et al., *Guillain-Barre Syndrome Following Vaccination in the National Influenza Immunization Program, United States 1976-1977*, 110 Am. J. Epidemiology 105-123 (2003), filed as "Pet. Ex. 35".

Dr. Steinman referenced a textbook by Dr. Donofrio on peripheral neuropathy that states children and adults with GBS present with rapidly evolving symmetrical muscle weakness within a matter of hours to just a few days. Tr. 189; Pet. Ex. 73 at 2.[146] He stated *Schonberger*[147] and *Donofrio*[148] do not discuss whether a recall response is involved with those respective onset timelines. Tr. 191. Therefore, it is his opinion that onset can occur that quickly, and timing in this case is not impacted if onset is found to be one day or three weeks, adding petitioner suffered from diabetes and pain prior to the vaccine but it was not until his receipt of the flu shot that he experienced "this terrible downward course." Tr. 191-92. Dr. Steinman agreed that date of onset and date of diagnosis are two different things, comparing it to cancer and saying that cancer begins when the first cancer cell went "haywire," not when a lump is found. CIDP is similar, so he prefers a three-week onset as supported by *Schonberger*. Tr. 246. After much posturing, he further agreed that the latency period between inciting event to onset of condition is different than the period between onset and nadir, the peak of symptoms after onset. Tr. 246-48. He agreed the *Donofrio* text was discussing the period from onset to nadir. Tr. 254.

However, he added, if this case is deemed a significant aggravation in light of petitioner's diabetic neuropathy, then the onset from the flu vaccine could be as early as a day or three to four weeks later. Tr. 231.

In Dr. Chaudhry's opinion, the medical records support a rather acute onset the day after the flu shot. While the Table says 3 to 42 days for GBS, it does not acknowledge CIDP. Dr. Chaudhry does not believe petitioner suffers from CIPD and therefore, the vaccine is not involved. Tr. 322-23, 524, 535; Pet. Ex. 13 at 9, 28.

a. Analysis of Prong III

Dr. Steinman vacillated on onset, refusing to commit to one day or three weeks, expressing a preference for three weeks but submitting that one day works just as well. The history petitioner provided to his treaters in his initial presentations to Dr. Hopkins on September 17, 2013 and to Dr. Tandon in the emergency room on September 29, 2013 are compelling. He reported onset of right leg pain the day after vaccination and an inability to return to work for 2-3 days, with symptoms that progressed and worsened over the next several days despite the use of over-the-counter medications, ice, and heat. Pet. Ex. 9 at 11-13; Pet. Ex. 3 at 8-11. Clearly, his presentation to the emergency room on September 29, 2013, forty-one days after his flu vaccination, with complaints of progressive, worsening symptoms since receipt of the vaccine showed a course of symptoms that became severe. His testimony of his return to work without problems, his ability to drive to the car show and his symptoms there, and his return to work thereafter until he was unable pick up coins or climb a ladder resulting in an emergency room visit are consistent with the waxing and waning of CIDP and explain why he felt better when Dr. Hopkins prescribed steroids on September 17, 2013. The record supports onset the day after petitioner received his flu vaccine on August 19, 2013.

[146] PETER D. DONOFRIO, TEXTBOOK OF PERIPHERAL NEUROPATHY 347, 350 (2012), filed as "Pet. Ex. 73".
[147] Schonberger et al., *supra* note 145.
[148] DONOFRIO, *supra* note 146.

According to Dr. Steinman, *Schonberger*[149] and *Langmuir*[150] support one month as an appropriate time frame for onset between vaccination and inflammatory neuropathy that is initially acute and evolves into CIDP. However, Dr. Steinman disregards that petitioner's presentation for medical care and GBS diagnosis one month after the vaccine was not when petitioner reported to medical personnel that his symptoms began. In fact, petitioner consistently reported onset the day after receipt of the flu vaccine at his first visit to Dr. Hopkins,[151] his first emergency room presentation,[152] and even when he presented to Dr. Xiong in September 2018, providing onset as when he awoke the morning after his vaccine.[153]

Where discrepancy exists between what the petitioner tells treaters and what petitioner testifies to at hearing, the undersigned believes and finds more credible the earlier contemporaneous medical records when petitioner was seeking treatment. Well-established case law provides that contemporaneous medical records are more believable than that produced years later at trial. *United States v. United States Gypsum Co*., 333 U.S. 364, 396 (1948); *Burns v. Sec'y of HHS*, 3 F.3d 415 (Fed. Cir. 1993); *Ware v. Sec'y of HHS*, 28 Fed. Cl. 716, 719 (1993); *Estate of Arrowood v. Sec'y of HHS*, 28 Fed. Cl. 453 (1993); *Murphy v. Sec'y of HHS*, 23 Cl. Ct. 726, 733 (1991), aff'd, 968 F.2d 1226 (Fed. Cir.), cert. denied sub nom. *Murphy v. Sullivan*, 113 S. Ct. 263 (1992); *Montgomery Coca-Cola Bottling Co. v. United States*, 615 F.2d 1318, 1328 (1980). Contemporaneous medical records are considered trustworthy because they contain information necessary to make diagnoses and determine appropriate treatment:

> Medical records, in general, warrant consideration as trustworthy evidence. The records contain information supplied to or by health professionals to facilitate diagnosis and treatment of medical conditions. With proper treatment hanging in the balance, accuracy has an extra premium. These records are also generally contemporaneous to the medical events.

Cucuras v. Sec'y of HHS, 993 F.2d 1525, 1528 (Fed. Cir. 1993).

The records are replete with references to the onset of petitioner's symptoms within a day of receipt of his flu vaccine. As stated by Dr. Steinman, petitioner's uncontrolled Type 2 diabetes with diabetic neuropathy, along with his other chronic and pre-existing conditions, worked synergistically with his receipt of the flu vaccine resulting in a decline of his already tenuous health, *Langmuir*[154] and *Schonberger*[155] support onset of an immune-mediated inflammatory reaction in the first two days following receipt of a vaccine, particularly when the individual has had previous flu vaccines, as was the case here.

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[149] Shonberger et al., *supra* note 145.

[150] Alexander D. Langmuir et al., *An Epidemiologic and Clinical Evaluation of Guillain-Barre Syndrome Reported In Association with the Administration of Swine Influenza Vaccines*, 119 AM. J. EPIDEMIOLOGY 841 (1984), filed as "Pet. Ex. 52."

[151] The record from petitioner's September 17, 2013 visit with Dr. Hopkins states he "woke up with right leg pain and weakness" after receiving the flu shot at CVS Pharmacy. Pet. Ex. 9 at 11.

[152] Petitioner reported "debilitating joint pain throughout body starting a day after he received a flu shot 9-19-13" at his first visit to the emergency room on September 29, 2013. Pet. Ex. 3 at 2.

[153] Petitioner reported to Dr. Xiong that his "arm or hand was painful" after receipt of the flu vaccine and the "next day, his hip joints hurt when he went to his car". Pet. Ex. 65 at 13.

[154] Langmuir et al., *supra* note 150.

[155] Shonberger et al., *supra* note 145.

Accordingly, petitioner has presented preponderant evidence to support Prong III.

4. Burden Shifting: Alternative Cause

a. Respondent Did Not Provide an Alternative Cause of Injury

Because petitioner has established a prima facie case of causation under *Althen*, he is entitled to compensation unless respondent can show by a preponderance of the evidence that petitioner's injury was in fact caused by a factor unrelated to the vaccine. *Deribeaux*, 717 F.3d at 1367; *see* § 13(a)(1)(B). To meet this standard, respondent must "present sufficient evidence to prove that the alternative factor was the sole substantial factor in bringing about the injury." *Deribeaux*, 717 F.3d at 1367. The Vaccine Act limits the scope of unrelated factors by excluding any "idiopathic, unexplained, unknown, hypothetical or undocumentable cause, factor, injury, illness or condition." § 13(a)(2)(A). "In other words, alternative causes that are 'idiopathic, unexplained, unknown, hypothetical or undocumentable' cannot overcome a petitioner's prima facie case." *Doe*, 601 F.3d at 1357 (quoting § 13(a)(2)(A)).

Respondent has not provided an alternative diagnosis, other than to argue that petitioner does not have GBS, CIDP, or CIDP as a result of his diabetes. This argument is insufficient to sustain respondent's burden.

VII. Conclusion

Upon careful evaluation of all the evidence submitted in this matter—the medical records, the testimony of petitioner and the experts, and the experts' opinions and medical literature—I find that petitioner has shown that he is entitled to compensation under the Vaccine Act related to his probable CIDP. Accordingly, this matter shall proceed to damages.

IT IS SO ORDERED.

s/ Mindy Michaels Roth
Mindy Michaels Roth
Special Master